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TREATMENT OF FRACTURES IN A CASUALTY HOSPITAL.[1]

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WHEN I was first asked to take part in the subject matter of this evening's meeting I was allotted the task of the treatment of fractures from a general practitioner's point of view. However, as I have had most of my experience of fractures inside a casualty hospital, it was decided to describe this paper as "The Treatment of Fractures in a Casualty Hospital." This to a great extent is the same as the treatment from the aspect of a general practitioner, as the "initial care" of fractures except in a large general hospital with its elaborate appliances resolves itself into the treatment under more or less glorified first-aid principles by a general practitioner. I have designated this as the "initial care," but it may be permanent inasmuch as the practitioner may find that after a few days the result is more than at first expected.

A first-aid or ambulance man is taught to attend to a fracture on the spot and to set it in such a way as to make further damage impossible until the arrival of a doctor or until the patient is transferred to one. The practitioner goes a step further and renders his first-aid treatment in a careful truly surgical way. He aims at placing the patient in a comfortable and safe position, to treat shock and minimize pain and discomfort. It is useless to attempt to set a bad compound fracture of the thigh if the patient is suffering severely from pain and shock. If there is no severe hæmorrhage it suffices to treat the wound quickly and antiseptically, to place the limb in as comfortable a position as possible and to administer a medium dose of morphine and perhaps strychnine or pituitrin, promote warmth by covering the patient with blankets and pack the bed with hot water bottles.

During my ten and a half years as Medical Officer at the Port Adelaide Casualty Hospital it has been my fortune to treat six hundred fractures and it is my intention this evening to give you an epitome of the experience gained from the work.

General Treatment of Fractures.

When a patient is admitted to the Casualty Hospital, where I might state there is no resident medi-

[1] Read at a meeting of the South Australian Branch of the British Medical Association on February 25, 1926.

cal officer, he is placed upon a firm fracture bed and if the fracture is of any extent, 0·016 gramme (a quarter of a grain) of morphine is administered, the limb put at rest on a pillow or between sandbags and the patient covered up with blankets. No attempt is made to make the patient clean, no matter how dirty he is, if his condition does not warrant it. If, however, the individual is not much distressed, the injured limb is alone uncovered, washed with soap and water and powdered, placed between sandbags and covered up pending my arrival.

The advantages of the immediate dose of morphine are that the pain is lessened or controlled, shock, restlessness and anxiety are minimized and last but not least muscular spasm and rigidity are relaxed, so much so that frequently I have been able to set an overlapping fracture of both bones of the leg without a general anæsthetic, even when strong traction has been needed.

If this is not possible I wait for a reasonable time to enable the patient's stomach to be empty, perhaps three or four hours if he has only recently had a good meal and then a general anæsthetic is administered. Apart from the avoidance of the usual dangers of giving an anæsthetic on a full stomach, there is the additional advantage that the patient is not so liable to vomit; this with its concomitant exertion gives rise to further shock and pain in the treated limb as the unfortunate individual is coming out of his anæsthetic.

Anæsthesia.

While on this part of the subject I wish to make a few remarks on anæsthesia. I know the present day teaching with its antipathy to chloroform, but with the big burly individuals that I frequently have to treat, I find that the induction with chloroform is the most satisfactory. Ethyl chloride in such a patient seems to produce too much rigidity and the sequence of open ether is insufficient in many cases to overcome it. No doubt an alcoholic history has a great deal to do with this and it is obvious that, provided the patient is carefully watched, the anæsthetic which produces the least rigidity and struggling is to be preferred, especially when sharp ends of broken bones are in possible contact with important vessels or nerves.

After anæsthesia is produced and good flaccidity has been obtained, the limb is thoroughly washed and powdered (if not previously done as referred to already) and then set in as good a position as possible with good alignment fore and aft as well as laterally, extension or traction (which is a far preferable term) not being forgotten. Splints *et cetera* are applied with careful padding and bandaged on.

When the individual is out of his anæsthesia the remaining limbs and the trunk are washed and cleaned and the patient made generally comfortable.

I stated just now about setting the limb as well as possible under this anæsthetic. I mention this because in having no X ray plant at the Casualty Hospital I am like the outside general practitioner in a private home or most private hospitals. In a large general hospital the textbook advice of examining the fracture with X rays from the start can be followed out, but under the circumstances that I have just related, I maintain that it is better to try hard to set the limb properly, fix it and maintain it in good position and then transfer to a larger hospital or private radiologist for examination and to take a picture of the limb in splints. If the photograph reveals good position, the patient does not then require to have a second anæsthetic; if the fractured bone is not in quite good position, the picture will show how much displacement there is to recorrect and in what direction this is necessary. When the patient is under the second anæsthetic this can be well borne in mind.

The Use of X Rays.

With the present day industrial legislation the public has learned to value the X rays and I recommend that, even if you are satisfied with the correction of a fracture without X rays, get a picture taken, because you can then satisfy a court of law that you have made use of every available help if your result is not perfect in the eyes of the laity. The public looks for a cosmetic as well as a good functional result and should either of these not eventuate, it would be hard to convince a judge and jury that the best possible result under the circumstances was obtained, if the X rays had not been commissioned.

Compound Fractures.

With a large compound fracture if hæmorrhage is not severe (and on account of the causative crushing it rarely is so) the patient is covered up to promote warmth and minimize shock and 0·016 gramme (a quarter of a grain) of morphine is given hypodermically. The wound is covered with an iodized dressing but not bandaged firmly. I do this so as to promote oozing which will assist in the loosening of infective material in the wound. As soon as circumstances permit a general anæsthetic is administered and ordinary tincture of iodine is poured into the wound and the excess swabbed out. If large, the wound is partly sutured up, but never wholly, as a small tube or gauze wick is left in for drainage. A dressing is applied without a bandage. The limb is set in the usual way, the final bandage being used round wound and splints so that the former can be redressed without disturbing the fixation. A dose of one thousand five hundred units of antitetanic serum is administered, although in injuries among coal lumpers I find practically no sepsis because coal in itself is more or less sterile. Healed wounds often manifest evidence of coal dust which remains like gunpowder marks or tattoo. The same thing is noticeable in the lungs seen at *post mortem* examinations in quite old men who have worked among coal nearly all their lives. The thick deposits of coal in their lungs seem to have done them little or no harm.

Do everything possible for your patient from the beginning. Remember, as I stated before, that your initial setting may be perfect or at least as perfect

as possible under the circumstances. If you are transporting the patient to a hospital, aim at setting that fracture so that it need not have to be taken down until an X ray photograph reveals the want of improved position. Use light wooden splints that will allow the passage of X rays and in fractures above the elbow avoid metal hinges for the same reason.

Do not be frightened of the criticism by the surgeon to whom you are sending the case. He may possibly have done no better under the circumstances. A casualty hospital or a general practitioner does not possess all the appliances, carpenter's shop *et cetera* that are nowadays called into operation for the continued fixation of fractures; so do not be dismayed if a young house surgeon who is used to such appliances, does make rude remarks about an outside practitioner's use of more or less old fashioned methods. Many of us did not have the fortune through no fault of our own of being on active service abroad and naturally we stick to methods we have been taught and used with success. So if some of the procedures that I give you are as old as Adam, you will kindly pardon me, because I am convinced of their efficacy.

Initial Treatment and Transport of Special Fractures.

So much for the general treatment of fractures. Now I will pass on to a few details in connexion with the initial treatment and transport of special fractures.

Earlier in this paper I mentioned that I have had to deal with six hundred cases of fracture during the last ten and a half years.

This total includes:

Fracture of the skull	69 cases
Fracture of the spine	4 cases
Fracture of the sternum	4 cases
Fracture of the ribs	106 cases
Fracture of the nose	18 cases
Fracture of the lower jaw	18 cases
Fracture of the clavicle	50 cases
Fracture of the scapula	1 case
Fracture of the humerus	13 cases
Fracture of the radius (Colles)	10 cases
Fracture of the radius, shaft alone	2 cases
Fracture of the ulna, shaft alone	12 cases
Fracture of the radius and ulna	83 cases
Fracture of the metacarpus	9 cases
Fracture of the thumb	9 cases
Fracture of the fingers	34 cases
Fracture of the pelvis	4 cases
Fracture of the femur	33 cases
Fracture of the patella	5 cases
Fracture of the tibia alone	11 cases
Fracture of the fibula alone	14 cases
Fracture of the tibia and fibula	63 cases
Fracture of the ankle (Pott's *et cetera*)	14 cases
Fracture of the heel	3 cases
Fracture of the big toe	9 cases
Fracture of the toes	5 cases
Fracture of the metatarsus	6 cases
Total	600 cases

Of these five hundred and thirty-nine have been simple fractures and sixty-one compound.

These totals do not include the innumerable crushed fingers and toes that have had to be amputated or trimmed up at once.

Fractures of the Skull.

Fractures of the skull cause more anxiety than any other fractures on account of the concomitant injury to the cerebral tissues. Naturally several of my cases have been fatal as the exciting cause has been extreme. When the patient is admitted, he is kept quite quiet and in the case of a compound fracture the wound is cleansed in the usual way, the neighbourhood of the wound is shaved and if possible the whole head; but this latter is not usually done until the patient shows some sign of reaction. The nasal and buccal cavities are carefully cleansed of foreign matter and artificial teeth are removed. If bleeding *et cetera* occurs from the ear, the pinna and external meatus are cleansed with iodine and a light gauze and wool pad is applied; the meatus, of course, is not syringed or plugged. In shaving a head be very careful in the movement of a patient. In the case of one patient who, I remember, had concussion and an extensive scalp wound and cerebral irritability, I shaved the head and sutured the wound with the help of a light general anæsthesia. I learned to my dismay afterwards that the patient had a fracture through one of the cervical vertebræ and, although I might be excused for missing it at the start on account of the concomitant cerebral injury, I naturally wished that I had not turned the patient or lifted up his head to shave the occipital region. Of course he did not feel it at the time, but on slight regaining of consciousness he complained of pain in his neck. Do not forget to examine for a full bladder. A good nurse will remind you of the fact if the patient is not voiding urine under him; but in any case do not forget it yourself. With a cerebral or spinal injury a single overdistention of the bladder may lead to an enormous amount of trouble from atony apart from a nervous retention that would otherwise have recovered. If you are moving your patient to hospital, do not attempt it unless you are tolerably sure he will not die on the way. Sending a man to a surgeon for a decompression is useless, if the man is not going to reach him. Also do not send a patient with a cerebral injury in an ambulance, unless you have a competent nurse or relative to go as well; because if reaction occurs from concussion the vomit may choke the patient. If no help is available go with him yourself. Also remember the time honoured pitfall of a diagnosis of alcoholism when a grave cerebral lesion may coexist.

Fracture of the Spine.

In fracture of the spine do not overexamine; look for cardinal signs; move the patient on a rigid structure with plenty of help available.

In these cases do not attempt any reduction until the patient has been examined by X rays; but in the meantime watch the bladder and guard against pressure sores and hot water bottle burns from the very beginning.

Fracture of the Sternum.

Fractures of the sternum are rare. Treatment consists in rest on a firm bed, application of

strapping to limit thoracic breathing and expectant treatment of possible intrathoracic injury.

Fractures of the ribs are common and give rise to a great deal of discomfort. The treatment is classical, but with prognosis beware, as it takes several weeks for a labourer in some cases to be fit for duty, even if his recovery is not retarded by his receipt of ample sick pay. Complicated cases of fractured ribs are not very common, but look for signs of pneumothorax within twenty-four hours, a case of which I had recently to my sorrow in an alcoholic patient.

Fracture of Nose and Lower Jaw.

On fractures of the nose and jower jaw I will not dwell, as the initial treatment is well known to all.

Fracture of the Clavicle.

The treatment of fracture of the clavicle is also well known to you all. The result of your treatment is usually splendid functionally, but not cosmetically. There is nearly always some deformity on account of inability to keep the fractured ends in good apposition. In the treatment of this I have almost always used Sayre's method, sometimes varied with careful bandaging instead of strapping, but always using the principles underlying the method. Strapping in hot weather is abominable and if the patient is not a naughty restless child or an alcoholic I find that good bandaging is often efficacious. With a medium sized pad in the axilla I find the chief thing to do is to support the elbow; the pulling of it forwards to throw the shoulder back is not so important; the patient can often do that himself because he can walk erect with shoulders thrown back (a movement that usually takes place bilaterally). Over the strapping I always apply fairly wide flannelette bandages and in the process of bandaging I place a cotton wool pad over the seat of fracture and pass the bandage two or three times over it. This serves to keep the fragments in position to a certain extent, but it has other uses inasmuch as it keeps the patient's fingers and the prying eyes of criticizing friends and relatives off the actual temporary anatomical deformity.

Fracture of the Humerus.

In fractures of the upper end of the humerus my custom is to put a small powdered pad in the axilla and to bind the upper arm to the side of the thorax, with the forearm supported by a small arm sling. The elbow should not be supported. I do not attempt to set the fracture until it is examined by X rays, because if it is an impacted fracture of the anatomical neck more harm than good can result from manipulation. The condition may be complicated by a dislocation or a fracture through the glenoid. Fix the arm to the side as described, have it examined by X rays and with the X ray picture in your mind give an anæsthetic and do what is necessary. This sounds contradictory to what I said a few minutes ago about setting a fracture before it is examined by X rays, but with an arm it does not really inconvenience the patient to have it examined by X rays twice, whereas with a lower limb the transport of a patient to an X ray plant from his bed may be irksome and harmful.

Fracture of the lower end of the humerus is different. Give an anæsthetic and examine carefully. Avoid by all means the internal angular splint, because this presses on the internal condyle and laterally displaces the lower end of the humerus. Put the limb up in full flexion and supination, fixing the forearm to the upper arm by means of a figure of eight bandage across the gap. Do not use strapping on the first day unless you are prepared to cut it and tape it together again instead of pulling it off the arm. This cutting becomes necessary sometimes within twenty-four hours or the bandage has to be loosened because of swelling around the elbow joint. For the same reason do not pad the anticubital fossa, but only cleanse and powder it. The supination and flexion hold the fragments laterally and anteriorly, whereas the triceps prevents the lower end of the humerus from being displayed backwards.

Fracture of the Olecranon.

Fracture of the olecranon before it comes to open surgical operation, should be treated by carrying the forearm in a large arm sling. It would seem feasible to fix the arm in full extension at the elbow, but this is inadvisable on account of the swelling caused by keeping the forearm hanging down.

Fracture of the Forearm.

In fractures of both bones of the forearm be careful to have the splints wider than the limb. If need be make a splint of thin flat wood and fashion it to the sound arm. You can then make it of the right shape and by reversing it fit it to the broken limb. The lower splint should be an internal angular, so as to fix the elbow and the distal end of it should be long enough to control the wrist joint to the palm. I do not mean that this should be permanent, but I certainly think the elbow and wrist should be controlled for a few days until the muscles have accommodated themselves and spasm is relaxed. A Colles fracture I still treat with an anæsthetic if necessary and fix on a Carr splint with or without a top splint. I do not use a "cock-up" splint in the early treatment of Colles's fracture.

Fracture of the Metacarpus.

Fractures of the metacarpal bones are by no means rare. I usually place a roller bandage in the palm (transversely), get the patient to grasp it and with a dorsal pad of wool I bandage the fingers well flexed over the palmar roller pad, taking especial care to prevent the metacarpo-phalangeal knuckle of the fractured bone from dropping.

Fracture of the Pelvis.

Fracture of the pelvis needs no special comment except that if the patient shows no sign of fractured thigh and active or passive movements of the legs give rise to pain in the buttocks or groin or over the sacro-iliac synchondroses, suspect fracture of the pelvis and keep him under observation and examine him with X rays.

Fracture of the Femur.

Fractures of the lower limb give the practitioner much anxiety both as far as the patient himself is concerned and also as regards the medico-legal relations between employer and employee. I fix all fractured femora for transport with a long Liston splint applied with a general anæsthetic. Unfortunately I have no experience with a Thomas splint for transport in these cases. My patients tell me that transport in an ambulance with a Liston splint leaves very little to be desired, especially as they have had an initial dose of morphine. Then again a long Liston is easily made and can be cut to any length within a few minutes. Under the anæsthetic I apply the Liston with a good axillary pad; fix the upper part to the chest with a binder and also the pelvis with a binder. Extension or traction is made by fixing the foot to the splint by means of the bandages passing through the prongs and by fixing the knee above and below the patella with bandages to the splint. This fixing to the knee provides the necessary extension or traction needed to overcome or minimize overlapping and I find no reason to use the perineal pad as the upper end of the traction takes place from the pad in the axilla and the binder round the chest. Of course, this is only for transport and I would not think of keeping a Liston splint on once the patient is in a hospital where he can remain for weeks and have extension and other elaborate apparatus applied.

Fracture of the Leg.

Last but not least I come to fractures of the leg, of which I have seen just over a hundred. I usually give an anæsthetic if the morphine has not sufficed and make strong traction on the lower fragment with the anæsthetist or nurse pulling above and the patient's knee slightly flexed. When the bones are in fairly good position I place the limb on a back splint which extends a few inches above the knee. At the lower end is an adjustable footpiece which is at right angles to the main part of the splint. Rotation of the foot is prevented by bandaging the foot to this footpiece and extension or traction is maintained by placing a figure of eight bandage above and below the knee and crossing behind over a small transverse piece fixed to the back of the splint just under the popliteal fossa. If possible I avoid Cline splints at the beginning except as an adjunct to the back splint. Cline splints during the first twenty-four hours give a lot of trouble, because it is hard to pad them properly over the bony prominences; secondly, the limb is liable to swell and cause bad pressure symptoms through the leg being in a more or less closed box and thirdly, the laterally placed splints prevent changing of a dressing over an oozing wound in the case of a compound fracture. Watch the heel; it causes a great deal of discomfort and in the case of Pott's or other fracture through the ankle joint a great deal depends upon the position of the heel as to whether the astragalus is in good position or not. This has especial importance on account of the ultimate functional result.

Conclusion.

In conclusion I must apologize for giving you treatments which are in no way new, but have stood the test of time; they have been practised by me for over ten years and I see no reason to change, although I have every opportunity and much material to work upon. I trust that I have refreshed the memory of many who do not have the fortune (or shall I say the misfortune) to see many fractures and I hope that I have made at least one point clear and that is: "Treat your fracture from the very start with the idea of obtaining a good anatomical and especially a good functional result."

TREATMENT OF FRACTURES OF THE LOWER EXTREMITY.[1]

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THE majority of those here have occasion at some time to treat a fracture of the lower limb. The principles upon which treatment is based, are now well established and you will realize that I feel I can add little in the way of novelty to a well traversed subject. I will remind you of those principles and emphasize the particular needs of some of the commoner fractures of the lower extremity.

In treatment we aim at union, at union with a full length limb, in good alignment. The simpler the means by which these can be attained, the better.

Fracture of the Femur.

An inch of shortening in the arm may give no trouble at all except to the patient's tailor, but if a fracture of the femur results in an inch of shortening, the patient will be branded with a limp for the rest of his life. And in almost every fracture of the femur there is this tendency to shortening which has to be combated. It is necessary to apply extension and the earlier in the case the extension is applied, the better the effect. There is no question to my mind that the simplest way of applying extension is by means of the Thomas splint. The Thomas splint can well be used in the treatment of any fracture of the femur from the very beginning; for compound fractures requiring a dressing it is essential. I would like to see the medical man in every country town in possession of one average sized Thomas splint. If occasion should arise when an out size is required, an intelligent motor mechanic at the local garage could readily make a second with the pattern to guide him.

One still occasionally sees a patient brought to the hospital with a long Liston splint applied for his fractured femur. This is a difficult splint to apply efficiently and when applied it is nearly useless; it provides no adequate extension.

[1] Read at a meeting of the South Australian Branch of the British Medical Association on February 25, 1926.

The war taught us that once the patient has reached an improvised stretcher, a Thomas splint can be applied, over the clothes if need be, and with a temporary extension made by means of a hitch round the patient's boot. In that way shortening is prevented from the beginning and transport made infinitely more comfortable.

During the first few days of treatment the measuring tape should be in frequent requisition. If shortening is allowed to persist beyond a week, it will be found increasingly difficult to overcome.

The other point to attend to is the alignment of the fragments. The limb lies in the Thomas splint free of bandages and in a fracture of the shaft of the femur the alignment can be gauged by an inspection of the shape of the thigh. I try to teach my students and myself to stand off and regard the general contour of a fractured limb. This applies with at least equal force to fractures of the leg below the knee. And present day fashion in dress offers no excuse to any who have not developed an artistic sense for the form of the normal leg. I would remind you that the alignment of a broken thigh has to be considered from the lateral view as well as antero-posteriorly. The normal femur has a considerably forward convexity. If the thigh looks flat, the bone ends have been allowed to drop backwards. An X ray picture taken in the lateral plane is the test, but a frequent inspection of the forward bulge of the thigh will give ready warning.

Fracture of the Neck of the Femur.

Fractures of the neck of the femur are to be treated with extension to prevent overlap (shortening), combined with full abduction to prevent *coxa vara* deformity which also would result in shortening. Apply a Thomas splint, tie the extension cord to the end, then swing the end of the splint right out over the side of the bed and attach it to the upright of a Balkan frame. A post screwed to the floor with a couple of fifteen centimetre (six inch) metal brackets will serve the purpose well. Treated in this way, these fractures even in elderly folk almost always will unite. The impression still seems to prevail that fractures of the neck of the femur do not usually unite. Such teaching has had a pernicious influence. If we aim at union, we get it. I have treated a series of cases of this fracture in old people by Hamilton Russell's method of extension in which no rigid splint is used at all. I have endeavoured to ascertain the opinion of experienced nurses about it and find that some prefer it to the Thomas splint for these cases, while some do not. So my own preference reverts to the splint. I would not recommend Hamilton Russell's plan as an alternative in the treatment of fractures of the shaft of the femur, as in inexperienced hands it permits a greatly increased risk of backward angulation.

Leg Fractures.

In fractures of the lower segment of the limb shortening and overlap do not occur to the same extent as is the case with thigh fractures and cannot occur to any appreciable degree unless both bones are broken. If an X ray film shows 2·5 centimetres (an inch) of overlapping in a fractured tibia, there must be a fracture somewhere in the fibula as well. Probably it will be found towards the knee, if another X ray picture is taken higher up.

Those who are accustomed to see many radiographs may smile at this self-evident fact, but one has seen cases in which apparently its significance is not realized. However, it matters little to treatment; if there is overlapping, extension will be necessary. We use then a Thomas splint with the extension cords attached to the ends of a crossbar of a wooden footpiece which is strapped to the foot. The typical Sinclair footpiece is rather more complicated than this.

In some quarters there is a tendency to use wiring or plating more readily in the treatment of leg fractures than elsewhere. The subcutaneous tibia may be tempting, but one cannot refrain from expressing the opinion that the use of bone plates by the occasional operator is strongly to be deprecated. The operation requires some degree of technical skill and familiarity with the use of carpenter's tools, it is seldom an easy one and, if the fracture is a few weeks old when plating is undertaken, the difficulties will be great. Rarely we meet with a case in which the application of a plate may serve better than any other method, but in the vast majority of cases intelligent perseverance with other methods will be rewarded with a better end result. An exasperated insurance company recently referred to me two cases in which plates had been used apparently for compound fractures about a year before. Each patient had a grossly septic area communicating with a metal plate.

Transverse fracture of the patella provides an exception and here the routine treatment is operative. But transverse fracture of the patella is to be regarded almost as a ruptured tendon and treated as such by suturing. The simplest effective way of suturing the ruptured *quadriceps* tendon is to wire together the two fragments of the patella incorporated in its substance.

As a contrast one may take the usual stellate fracture of the patella, in which no tendinous separation occurs and which is, therefore, to be treated without operation. I can show you also the X ray pictures of a transverse fracture of the patella in which the tendinous fibres of the *quadriceps* evidently remained intact. There was no tendency to separation of the fragments even when the knee was flexed. This man was, therefore, treated without operation, indeed without any splint at all. He stayed in bed for a week or two and was permitted to move the knee gently from the beginning. He is now back at work with perfect function and full range of movement.

As is well known, the lower third of the leg is one of the most likely situations for a fracture to fail to unite. The treatment of non-union is not the occasion for the use of a metal plate, but rather for a bone graft. I prefer to use a sliding graft taken from the same tibia above the fracture.

Fractures of the shaft of the tibia alone require no extension and are usually best treated by some form of back splint. This skeleton (Crab) splint is more secure than the ordinary wooden back splint and it may be bent at the knee if required. With a single piece of adhesive plaster encircling the thigh and upper end of the splint, all other bandages may be removed for inspection or for purposes of massage without the position of the leg being disturbed at all. I think it a good thing to have few bandages as possible covering a fractured limb. One of the advantages of a Thomas splint is that it can be used with no covering bandages at all. The same might almost be said of this splint. If a limb is swathed with wool and bandages, it is not likely to be inspected as frequently as it should be. If it is bare, one can take in the whole limb at a glance and instantly appreciate any lack of symmetry. Massage too can be more readily applied. In leg fractures it is undoubtedly of benefit if gentle massage is instituted almost from the beginning. Massage to some extent compensates for the stasis of lymph flow which inevitably occurs in a fractured limb, coagulation is prevented and and we do not then see that firm wooden œdema of the limb. The massage I order is intended to do this only—passive movement of joints is not permitted.

Pott's Fracture.

One more common fracture I would allude to, that is Pott's fracture. Pott's fracture is to be regarded as primarily a dislocation at the ankle joint, a dislocation of the foot outwards or out and back. The accident is usually a forcible wrenching of the foot in eversion, the thrust of the astragalus against the outer malleolus fractures the fibula a little higher up. The fibula being fractured, the foot dislocates outwards or backwards to a degree corresponding with the violence. If the internal malleolus is torn off by the pull of the internal lateral ligament of the ankle, displacement can occur the more readily.

If the condition is regarded in this way, clearly our first duty in treatment is to reduce the dislocation. Efficiently reduce the displacement and the broken end of fibula is dragged into position by the tethering ligaments. Dr. Smeaton has emphasized the importance of reduction in the treatment of Colles's fracture at the wrist. It is equally important in dealing with Pott's fracture first to reduce. No splint can be relied upon to push the ankle into position. A very simple splint will as a rule hold it in position, once you have got it there. Under an anæsthetic the foot is wrenched carefully into a position of inversion and is dorsiflexed. If there is backward displacement as well (shown by the projection forwards of the lower edge of the tibia) get an assistant to fix the leg against the bed while you lift the foot forwards by the heel. Then test dorsiflexion. Apply a back splint to keep the foot at right angles to the leg. Apply pads to keep the foot in slight inversion, one pad over the outer side of the foot and one on the inner side of the shin higher up. It is of great advantage to have a radiograph taken after this has been done.

In those less common cases in which after reduction displacement tends immediately to recur, it will often be found that a portion of the lip of the tibia has been broken off in addition to the usual Pott's fracture.

If union of Pott's fracture is allowed to occur with the foot in eversion, the mortice of the malleoli is permanently widened and the astragulus will be liable to wobble. Such a condition can also occur by bending of the soft new callus, if walking is permitted too early. When ready for walking the patient must wear a heeled shoe and the heel must be made eight millimetres (one-third of an inch) higher on the inner side, so as to tilt the foot into inversion and take strain off the broken fibula. I have emphasized these well known facts because one still sees patients, often compensation claimants, in whom lateral displacement or an even more crippling backward displacement has been allowed to persist. The trouble in most appears to be due to a lack of appreciation of the need for reduction of the displaced ankle.

Plaster of Paris has a place in the treatment of fractures of the leg. Used early there are risks, but a week or two later a light plaster casing is helpful in enabling the patient to be more readily moved. One or at most two of these gum and plaster bandages will make a casing extending from the foot to the knee. Stick a strip of motor tube rubber to the leg with a tiny piece of adhesive and bandage over that. In ten minutes or so run a sharp scalpel down the plaster, but do not divide it completely. Also score a line down the back with the scalpel. The remaining filaments in front may be divided next morning and the foot can be lifted out as desired.

One has seen cases of fractured sesamoid bones, of fracture of the *os calcis*—a student fractured his astragalus in swinging round to take a ball at lacrosse—and other rare fractures. I have dealt only with common ones which we all meet and of which our treatment might well be standardized.

NOTES ON FRACTURES AND DISLOCATIONS OF THE UPPER EXTREMITY.[1]

By BRONTE SMEATON, M.B., B.S. (Adelaide), M.R.C.S. (England),

Honorary Surgeon, Adelaide Hospital, Adelaide.

IT is my intention in the twenty minutes allowed me to refer only to those injuries which seem to me to require consideration not always given them in systematic works on surgery.

At the outset let me say that restoration of function rather than anatomical perfection is the object of treatment and that operative treatment is used rarely and only after failure of other methods.

Operation which involves the disturbance of tendons in their sheaths or beds, is often very dis-

[1] Read at a meeting of the South Australian Branch of the British Medical Association on February 25, 1926.

abling, although the bones are brought together. Such a success at the expense of function is to be avoided.

The clavicles are regarded as spreaders between the sternum and the acromion and fracture or dislocation allows of undue approximation of these structures. The supine position of the body corrects the deformity, since the shoulders fall back and the weight of the arm does not drag down the shoulder. Sayre's method has antiquity to recommend it, but I find it very little used by surgeons of experience —its mechanical intention is good, but difficult to accomplish without pain, as I will now demonstrate.

A band of strapping drawing the elbow to the dorso-lateral chest wall is usually enough and the patient's hand is left free. A dislocation of the clavicle from the acromion is managed by Jones's method with sticky pads and a bandage.

Fractures of the surgical neck of the humerus are treated with the arm abducted to the necessary degree. Sometimes a Middeldorf triangle is enough and sometimes a splint that places the humerus horizontally. The degree of elevation depends on the extent to which the muscles attached to the tuberosity of the humerus elevate the upper fragment. An injury that threatens ankylosis of the shoulder joint calls also for this position, as ankylosis in abduction permits useful function of the arm. The mobility of the scapula allows adduction, but not abduction in ankylosis.

Fractures of the shaft of the humerus should not be treated with extension; the tendency is for the fragments to separate rather than to overlap.

In fractures near the elbow joint full flexion is a suitable position for treatment except in the case of the olecranon. The displaced head of the radius may prevent this position and need removal.

The condition of *myositis ossificans* seems more likely to occur in fractures of the lower end of the humerus than elsewhere. Too early or too much movement seems to induce this complication. Dislocation of the head of the radius from the orbicular ligament is often overlooked and it is only when the fat child is older and thinner that the radius is found bearing on the outer condyle instead of on the capitellum. This deformity was found frequently in examining recruits.

Fracture of both bones of the forearm at the same level is an injury that causes anxiety. The surgeon knows that approximation of the four fractured ends or the angulation inwards of either bone may interfere with rotation of the hand. Here one may be content if reduction secures the separation of the bones even though there be angulation away from the midline of the arm of one or both bones. Such a position has a disturbing appearance when examined by the screen, but will result in a useful and shapely arm. Great stress was laid by some teachers recently on the action of the muscles of pronation in such fractures, but practice does not support the theories enunciated.

Splints of greater width than the limb or perhaps a "sticky pad" laid lengthways between the bones on the back of the arm will secure separation of the bones. It is wise to use splints which reach well down the fingers to obtain the firmest fixation possible.

Colles's fracture is so often undetected or reduced imperfectly that no apology is needed for speaking of it. Before immobilizing a Colles fracture one should see that full flexion of the wrist is possible and feel the little concavity on the front of the radius 1·25 centimetres (half an inch) above its extremity. After manipulation these tests should be made to insure that reduction is complete.

Flexion before reduction is prevented by the projection of the upper fragment into the hinge of the joint and by the tension of the extensor tendons over the sharp edge of the lower fragment.

Robert Jones's method of reduction is good and easy.

It is his custom to place the limb in a straight splint, back and front, extending well down the fingers. Others apply strapping with the wrist in full flexion. This method has the advantage of determining that the reduction is complete.

Fracture dislocation of the proximal row of carpal bones from the radius is difficult and sometimes impossible to reduce and maintain in reduction.

Removal of the trapezium and trapezoid and *os magnum* leaves a useful wrist and the only function that is impaired is the movement of the hand from side to side.

The metacarpal bones can be safely treated by strapping the hand over a rolled bandage in the palm. The usefulness of the hand of the pugilist with an additional row of knuckles proves that angulation, though inelegant, is harmless as regards function.

Reports of Cases.

ADIPOSIS DOLOROSA IN TWO CASES.[1]

By F. S. HONE, M.B., B.S. (Adelaide),
Honorary Physician, Adelaide Hospital, Adelaide.

MRS. W., aged sixty-five years, was admitted on November 30, 1925, for pain in the upper part of abdomen on left side. It was worse after meals and accompanied by a sense of fulness and belching up wind. The pain was described as a heavy dragging pain. She was said to have suffered from indigestion for years and was sent in as possibly suffering from carcinoma of the stomach. She was said to have lost four kilograms (nine pounds) in weight in the previous month. There had been no vomiting. The appetite was fair.

Ordinary physical examination revealed nothing in the digestive tract to account for the pain, but she was found to have large, rather localized deposits of fat which formed distinct tumours round the hips, on the insides of the thighs and just inside the knees. These deposits continued down the legs, but stopped abruptly just above the ankles; the feet were small. The masses of fat were firm and tense. All these deposits were distinctly painful on being

[1] Read at a meeting of the South Australian Branch of the British Medical Association on February 25, 1926.

picked up. A similar lesser deposit existed as a pendulous fold of fat across the lower part of the abdomen and this also was tender. There was an excess of fat on the posterior surface of the upper part of the arms, but none in the supraclavicular regions, on the face or backs of the hands; the hands were relatively small like the feet.

There was also a very definite scaly condition of the skin of the legs, especially noticeable on the shins; the skin of the abdomen and arms was dry; the hair on the head was dry and had fallen out slightly. The axillary hair was absent, pubic hair was rather scanty and eyebrows were present.

Menstrual history had been uneventful. She had married at twenty-three years of age, had had five children and had ceased childbearing at thirty-one years of age. The menopause came on at forty-eight years of age. The dryness and scaliness of the skin had been present from childhood. Some fatty deposits had occurred subsequent to cessation of childbearing at thirty-one years of age, but the most rapid development, especially on the hips and legs, had occurred since the menopause.

Subsequent investigations showed that the basal metabolism was more than 20% below normal. Her sugar tolerance was increased; her fasting blood sugar was 0·1% and did not rise about 0·14% after fifty grammes of glucose. She gained weight during the first week in hospital. There was no mental disturbance. Her temperature was not definitely subnormal.

The condition thus seemed to be a typical instance of *adiposis dolorosa*. The interesting thing was the part apparently played by several glands in causation of the condition. The scaliness of the skin from early life is usually ascribed to thyreoid deficiency. The way in which obesity had come on later in life, is generally put down to pituitary insufficiency and the rapid increase of fatty deposits after the menopause coupled with the excessive trochanteric deposits (usually associated with gonad insufficiency) suggested that these glands also had some part in the production of the syndrome. The history, however, suggested that the thyreoid was primarily at fault and X ray examination of the skull showed a normal *sella turcica*.

She was, therefore, put on dried extract of thyreoid gland in doses which were increased until they reached fifty grammes (twenty grains) a day. She lost about 0·9 kilogram (two pounds) a week on this treatment and the skin gradually became less scaly and the subcutaneous deposits of fat much softer.

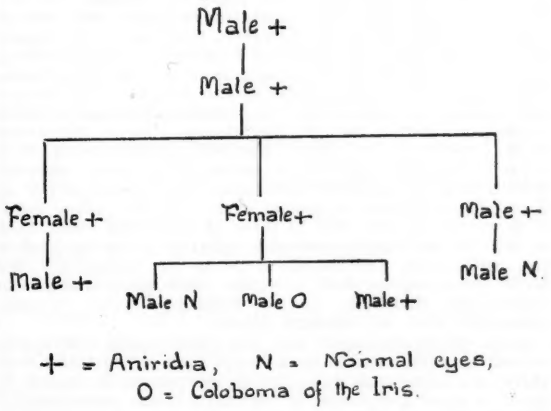
Her attacks of abdominal pain recurred every few days and as a fractional test meal done on December 3 had revealed a low gastric acidity with a weak chemical reaction from occult blood and as the X ray picture of the intestinal tract after a barium meal on December 7 had revealed a questionable penetrating ulcer on the posterior wall of the stomach, a surgeon was asked to see her at the beginning of January. An exploratory laparotomy was advised, but from various delays was not performed until February 4. At the operation no abnormality in the stomach or other abdominal regions could be found except that the omentum was heavily loaded with fat. The dose of thyreoid extract had been diminished to one gramme (fifteen grains) a day on January 8 owing to some intolerance and continued at this till the time of the operation. Recovery from operation was uneventful and thyreoid medication was resumed on her return to the medical ward on February 21. Notwithstanding the three weeks' intermission in medication the improvement in the condition of skin had continued, so that now the scaliness is almost gone except on the left leg. She is feeling much better and brighter than on admission, has lost nine kilograms (twenty pounds) altogether and is able to get about better, except for weakness following the operation.

One interesting point is that the sister of this patient has the same complaint only in a worse degree, in that she has suffered much more pain in the fat deposits. I managed to see her when she was visiting my patient in the hospital today, but she refused to come to the meeting tonight. The sister who is sixty-nine years of age, also ceased childbearing at thirty-one years of age. After this the fatty deposits were first noticed. They were quite definite at forty years of age. She went to England for special treatment in 1913. The pains were very bad until about five years ago, so bad that she says that at one period she thought she would go out of her mind. She has never had the scaly skin of this patient. The fatty deposits on the legs are the same and in the same situation. The abdominal and deltoid folds are much more prominent and there is a more general deposit of adipose tissue over the whole body than in this patient. The axillary hair is absent, the pubic hair somewhat scanty, but the eyebrows and hair on the scalp normal for a woman of her age.

ANIRIDIA IN FOUR GENERATIONS.

By J. BROOK LEWIS, M.B., B.S. (Melbourne),
Honorary Assistant Ophthalmologist, Adelaide Hospital; Honorary Ophthalmologist, Children's Hospital, Adelaide.

H.B., a male, aged six years, was brought to the Out-Patient Department of the Adelaide Children's Hospital because of defective sight. He had double aniridia and horizontal nystagmus. His refractive error was + 6 diopters in the right eye and + 7 in the left eye in both meridians. The mother who brought the child, had double aniridia, vertical nystagmus and definite diffuse lens opacities in both eyes. The correction of her glasses was + 1·75 spherical lens for the right eye and a one diopter + spherical lens for the left eye. The mother's father and grandfather were affected as were also a sister and brother. The family tree as far as could be ascertained is shown in the accompanying diagram.



Of the two adult females examined one had had both lenses removed for cataract, the left with a disastrous result. The other female had diffuse opacities in both lenses. This is in accordance with what is usually found in cases of aniridia, the lenses suffering change at an early age. One child had coloboma of the iris, as if the absence of the cells in the embryo was in his case only partial. Another point was the absence of complaint. Only in bright light did they appreciate the disadvantage of their condition. Parsons states that the influence of heredity is seen in this abnormality more than in other ocular malformation. Thus Guthier found ten cases in four generations, Galazowski thirty-one cases in three generations, de Beck seven cases of aniridia and two of coloboma in three generations and Mohn found a mother with complete and two sons with partial aniridia.

Reviews.

OSLER'S MEDICINE.

THE appearance of the first volume of a third edition of Osler's "Modern Medicine" under the editorship of Thomas McCrae calls for more than passing comment.[1] Probably the first thought that will come into every reader's mind will be the sorrowful reflection that Osler, the originator of the work, is no longer with us. All will applaud the sentiment that led to the reprinting in place of a preface of the delightful essay on the evolution of internal medicine which William Osler wrote as an introduction to the first edition.

That first edition almost at once became recognized throughout the English speaking world as the standard reference book on medicine and though other systems have since appeared, Osler's system has never been displaced. Unfortunately from the point of view of its utility the rapid advances made in medical knowledge in these days render any system out of date in some details almost before it is out the printing press. For a time this is of course of little moment as the newer knowledge is accessible to all in the current medical journals, but the time soon comes when the mass of new information is so great that almost every article in the system has to be supplemented by extracts from more recent writings. When we read that it is twelve years since the first volume of the second edition saw light, we realize that the time is fully ripe for revision.

The volume under review deals with infectious diseases and the editor has shown excellent judgement in his choice of twenty-three contributors from both sides of the Atlantic.

It is obviously impossible in a review of a book of more than eight hundred pages to do more than give a very general opinion, but we have no hesitation in saying that every article is a good one and that several are excellent. No one will regret having added the book to his library. A few articles may be specially mentioned.

Typhoid fever is dealt with in an admirable monograph of one hundred and seventeen pages by Thomas McCrae. It is so good that one hesitates to offer even minor criticism. We do think, however, that too much insistence is laid upon the danger of diagnosing a complicating appendicitis when there is pain in the right iliac fossa, especially in the early stages of the disease. The danger lies rather in regarding too complacently such pains vaguely as just a part of the disease. Every physician with experience will have handled patients with very acutely inflamed appendices which have been removed within the first ten days of the disease; the operation *per se* does not constitute a special risk. On the other hand it is worthy of note that in three of the thirty cases, that is 10% of perforation specially referred to by the author the perforation was found to be in the appendix. We were surprised to find febricula mentioned under the differential diagnosis as a definite disease, usually associated with an enlarged spleen.

It is disappointing to find the paratyphoid fevers dismissed in little more than a single page as we think that there are often sufficient clinical differences in regard to mode of onset and course to warrant a much more detailed account than is here supplied.

George Norris and David Farley contribute so complete an account of lobar pneumonia that it seems hypercritical to note that they make no mention of the liability of this disease to recur about areas of lung tissue involved in local disease, such as hydatid disease and new growths. Yet this is worth remembering, especially in this country.

Australians will be interested to read under the heading of seasonal causes of pneumonia that in Australia the fact that the highest death rate from this disease is in August and September is attributed to the exhausting heat and the fact that when the air is dry, it is a better carrier of microorganisms.

Two hundred and thirty pages are devoted to the best account of tuberculosis we have yet read. The chapter on the pathology of tuberculosis by Allen K. Krause is delightfully written and supplies abundant food for thought. Some, however,will feel that he is drawing rather a wide net when they read in his opening paragraph. "It has become second nature for us to think of everybody as the abode of living tubercle bacilli, of most of mankind engrafted with tubercle built round these bacilli, yet the greater part of these tainted beings scatheless and only a moiety prey to those same bacilli."

We note that in his chapter upon the history and ætiology Dr. Baldwin writes: "The introduction of corrosive gases during the Great War was expected to play a great part in the causes of post-war tuberculosis. Fortunately there have been few cases clearly due to these injuries," We understand that medical officers of the Australian Repatriation Department would question this statement.

Dr. Herrick's contribution on meningococcus infections appears to us perhaps the most perfect essay in the series. He selects this title in preference to the more usual name epidemic cerebro-spinal meningitis on the ground that the evidence all points to the conclusion that the lesion in the subarachnoid space is really a metastatic stage arrived at after the organism, originally implanted in the nasopharynx has entered the general circulation. It is obviously of great importance from the point of view of protection of both patient and those in contact with him that attention should be focussed upon the earlier phases of the disease rather than upon the final metastasis. The treatment is particularly well described and the reader is given very full and definite details of how the general disease and the various contingencies should be treated.

In some of the other articles this definiteness about treatment is rather lacking and this is a distinct defect. A system of this kind is regarded by the general practitioner as his standby in an emergency and we think that when he turns to it, he should find the plan of treatment regarded by the writer as the best, clearly and unmistakably laid down and that this should not be in any way confused with the various alternative therapeutic measures which should, of course, be also referred to in a work of this magnitude.

[1] "Modern Medicine its Theory and Practice," edited by Sir William Osler, Bart., M.D., F.R.S., Re-edited by Thomas McCrae, M.D., Assisted by Elmer H. Funk, M.D.; Volume I.: Bacterial Diseases, Non-Bacterial Fungus Infections, The Mycoses. 1925. Philadelphia: Lea and Febiger; Sydney: Angus and Robertson, Limited. Royal 8vo., pp. 877, with illustrations. Price 42s. net.

PSYCHOLOGY FOR NURSES.

"PSYCHOLOGY FOR NURSES" is a fair and eminently readable exposition of the new psychology as enunciated by Freud.[1] Miss Chadwicke is an ardent advocate and a whole-hearted believer in the Freudian doctrines which she occasionally pushes to extremes. For instance she says (page 8): "In the condition known as shock we find an excellent example of this occurrence (regression) in a superlative degree. Here we see a form of regression where the psyche is quite unable to keep pace with the rapidity with which it happens (*sic*) and where the gratification gained by it is connected with the unconscious death impulse. In its most profound form it involves loss of consciousness from which there is no recovery for the patient who passes on to the deepest regression of all, namely death which in a great many ways, which will be explained in a later lecture, may be regarded as an equivalent of the ante-natal state."

It is only just to state that the book does not contain many extravagances of this kind. On the other hand, while it omits nothing from the terminology, the interpretation of life and conduct or the mysticism which the more extreme adherents of Freudianism might desire, it is wholly free from those offensive traits which are often associated with the writings of many members of that school.

In the preface Miss Chadwicke states that the object of the lectures is not to train nurses to become psychoanalysts, but to ease the strain of nursing and to resolve those conflicts within the "unconscious" which, both in themselves and their patients, create friction. With such a laudable object few people will be inclined to quarrel.

[1] "Psychology for Nurses: Introductory Lectures for Nurses upon Psychology and Psychoanalysis," by Mary Chadwicke; 1925. London: William Heinemann (Medical Books), Limited. Crown 8vo., pp. 260. Price: 6s. net.

The Medical Journal of Australia

SATURDAY, MAY 1, 1926.

The Gordon Craig Fellowship in Urology.

The science of medicine has of recent years extended over such a wide field and the endeavours of those who would discover the hidden secrets of health and disease, have led into such intricate by-paths, that it is quite impossible for any one individual to have an adequate knowledge of its whole extent. Specialization has been not only inevitable but necessary. The explanation lies without doubt in the extension of biochemical and biophysical knowledge and in the results of bacteriological and immunological research. No medical practitioner, be he one who is dependent on his diagnostic skill and therapeutic acumen or on his manipulative dexterity, can afford to be ignorant of the why, how and wherefor of his scientific and laboratory aids any more than he can be prepared to dispense with their use. In no specialty is this truer than in urology. Urology, as we have previously pointed out, should embrace all diseases of the kidneys and urinary tract. Most urologists, however, limit their activities to those conditions which can be treated by surgical means. Even with this acceptation of the term competence as a urologist implies something more than skill in the use of the cystoscope. No mere surface knowledge of biochemistry is required for an accurate determination of the functionating power of a kidney. Discrimination and sound judgement are necessary for the proper interpretation of bacteriological and radiological findings and furthermore a urologist who essays to treat patients without possessing a sound knowledge of internal medicine at any rate as it affects the urinary system, will certainly come to grief.

Urology as a specialty has not received adequate recognition in Australia. The first urological department to be established in the Commonwealth was that started in 1914 at the Lewisham Hospital, Sydney. The pioneering work of Dr. Harry Harris at this clinic is well known to readers of The Medical Journal of Australia. In 1919 a department of urology was brought into being at the Royal Alexandra Hospital for Children, Sydney, and Mr. Gordon Craig was put in charge. The work done at this clinic was the basis of an important communication by Mr. Craig at the first session of the Australasian Medical Congress (British Medical Association) in Melbourne in 1924. With these two exceptions hospital boards have not until quite recently been alive to the advantages which accrue from intensive study of urinary diseases. It is true that at the Royal North Shore Hospital of Sydney a urologist was appointed some time ago, but for some reason best known to the Board of that institution the appointee was given the status merely of an assistant surgeon. Early in this year the Royal Prince Alfred Hospital of Sydney, the first teaching hospital in the Commonwealth to do so, determined to initiate a department of urology. Applications were called for a urological surgeon and Mr. Gordon Craig was appointed. The subject is of such importance from the point of view of both surgeon and patient that it is much to be desired that other teaching hospitals in Australia will follow the lead of the Royal Prince Alfred Hospital. Statistics of operative results published in Australia have shown conclusively that the percentage of recovery from urological procedures is much higher when the operation has been performed by a urological than by a general surgeon.

Following soon after the appointment of Mr. Gordon Craig to the charge of the new department at the Royal Prince Alfred Hospital has come the news of his magnificent gift to the University of Sydney in the provision of funds for the founding of fellowships in urology. Mr. Craig has equipped a laboratory in which urological research will be carried out. A fellow will be appointed and he will work for a period of three years. If at the end of that time the scheme is a success and there is no reason why it should not be, Mr. Craig has undertaken to bring his benefaction up to the handsome sum of £20,000. The gift is most generous and is the sincere expression of the wish of the donor to place urology on a sound footing in Australia.

We have drawn attention to the qualifications necessary in one who undertakes the exclusive practice of urology. It may be taken for granted that the fellows chosen under this scheme will eventually become practising urologists. It is desirable then that intending applicants for appointment to fellowships should realize that they will be required to possess all the attributes of a highly educated individual and to have had considerable experience in general and surgical practice.

Current Comment.

CHRONIC APPENDICITIS.

AN acute inflammatory process may terminate in several ways. It may end in resolution and the tissue may return apparently to its normal condition. On the other hand suppuration may occur and this is accompanied by tissue destruction. The destruction may take the form of pus formation, ulceration or sloughing or the whole mass of tissue may become gangrenous or necrotic. The acuteness of an inflammation depends mainly on the type and virulence of the infecting microorganism, but also on the reaction or "resistance" of the infected tissue. The whole inflammatory process is essentially one of repair. In acute inflammation the disturbance is mainly vascular and if the local vascular disturbance has not been extensive, little of a permanent nature may remain to mark the site of the process. In a more chronic type of inflammation there is a greater tendency to cell proliferation. This may be so extensive that tumour-like masses or granulomata are formed. In any case the end result is a condition of fibrosis. The best example of this type of chronic inflammation is probably a chronic tuberculous focus of long standing.

In inflammation of the appendix every variety of termination may be found. Fibrosis may be present to such an extent in the walls of the organ that its lumen is practically obliterated. This condition is as a rule the result of a series of attacks of inflammation of a subacute type. The occurrence of a chronic appendicitis has been questioned by many capable observers. In his useful article published in a recent issue of this journal, Dr. Rutherford Darling expressed the opinion that chronic appendicitis may be considered under three heads, recurrent appendicitis, relapsing appendicitis and chronic appendicitis proper in which the patient suffers from pain referred to the epigastric or umbilical rather than to the left iliac region, pressure over the appendix giving rise to the reflex epigastric pain. It would be better for the avoidance of all confusion if recurrent and relapsing appendicitis were not considered in the category of chronic appendicitis.

That opinions are divided on the subject of chronic appendicitis was abundantly shown at a recent meeting of the American Association of Obstetricians, Gynecologists and Abdominal Surgeons.[1] Dr. Arthur E. Hertzler for example holds that there is no such thing as chronic appendicitis. In an interesting paper he supports this view. The term inflammation implies a local reaction to an irritant, it implies a process and not a state. He holds that failure to keep this fact in mind is the fundamental error made by those who believe in chronic appendicitis. A terminal fibrosis is not a chronic inflammation. Surgeons do not distinguish a relapsing acute inflammation from an alleged chronic inflammation. An inflammatory process is made up of three stages or states, the acute reaction, the stage of repair and the terminal fibrosis. A chronic inflammation is a progressive hyperplastic process due to a slight but constant irritant. Acute inflammation is usually due to one of the pus-producing organisms, while a chronic inflammation is due to some other organism, such as that of tuberculosis, syphilis, actinomycosis and the like or to attenuated organisms of the pus-producing group; in other words it is a progressive chronic process. Dr. Hertzler then discusses the presence of the cells in the walls of the small intestine and in its mesentery independent of the lymphatic follicles. These cells are often spoken of as evidence of chronic inflammation. This in Dr. Hertzler's opinion is an error. They are found in all ages and in most animals. He shows that what is regarded as a chronic reaction process is nothing more than a variation of the normal. Dr. Hertzler, however, rather stultifies himself later in his article by admitting that in order to please his surgical chief, he, as pathologist, has frequently reported as evidence of chronic appendicitis the very changes which he declares are merely a variation of the normal. Be that as it may, he has provided food for serious thought. He concludes that as a pathologist he is utterly devoid of any knowledge that will clear up the problem of chronic appendicitis, but that as a clinician he is not so helpless. He has investigated the subsequent history of two thousand patients who had pain and tenderness in the region of the appendix and who did or did not have the appendix removed. He divides them into four classes: Those who are relieved of pain after removal of the appendix; those whose pain persists or returns after an intermission subsequent to removal of the appendix; those whose groin pains are relieved without the appendix being molested and in regard to whose condition there has occurred an error in diagnosis which becomes obvious later on. It will be remembered that Dr. Darling pointed out that almost without exception symptom-producing chronic appendicitis is associated with an anatomical condition interfering directly with the free drainage of the appendix. The lesion in his opinion is a mechanical one. He insisted that any operation for chronic appendicitis must be an

[1] *American Journal of Obstetrics and Gynecology*, February, 1926.

exploratory one, a short cut to diagnosis. Dr. Hertzler comes to very much the same conclusion, for he declares that the symptoms alleged to be due to chronic appendicitis can be relieved by searching for the actual cause and removing it.

Views of a different character to those of Dr. Hertzler were expressed at the same meeting by Dr. Robert T. Morris. Dr. Morris describes five different types of chronic appendicitis, two or them irritative and three infective types of lesion. The first type described by him is "an irritative lesion belonging to normal involution of the appendix." In this lesion all the structures of the appendix gradually undergo replacement by connective tissue with the exception of the peritoneal coat and the nerve elements. The latter become pinched and irritated in the course of contraction by the connective tissue. This is surely an *ex cathedra* statement. No evidence is offered in support of this "normal" process of involution. Why is not the appendix of every aged individual found *post mortem* to be in a fibrotic state. He mentions involution occurring in young people. If it occurs in young people, is it a normal process? In young people this fibrotic state of the appendix is held to signify a "stigma of physical decline." It may be accompanied by ptosis of the abdominal viscera, a loose kidney, crowded teeth and one or more of the stigmata of arrested development.

Dr. Morris's second irritative type of chronic appendicitis is the result of scar tissue following an attack of acute appendicitis. It appears possible that some of the cases in Dr.Morris's first group may in reality belong to his second group. The fibrotic state may be due to unrecognized attacks of a subacute nature. Moreover it is likely that some of the cases described by Dr. Morris as associated with ptosis of abdominal viscera, loose kidney and so forth are due to these conditions. Dr. Darling regards the symptoms of chronic appendicitis as due to interference with the drainage of the appendix from various causes. In a word the existence of Dr. Morris's normal involutionary process requires proof. The three infective types of chronic appendicitis are: (i.) the type associated with chronic inflammation of the neighbouring caecum and possibly with entozoa in the appendix; (ii.) a type characterized by lymphoid hyperplasia and (iii.) a type associated with chronic congestion of other parts of the bowel and relating to blood or lymph circulatory disturbances.

It is thus evident that the last word has not been written about chronic appendicitis. For the man who believes in the unnecessary type of what has been so aptly termed operative interference, the subject will present no difficulty until he is faced with a patient unrelieved of symptoms. In that case he will probably interfere again. The man who prefers to play for safety, will do one of two things. He may undertake surgical operation and plan his procedure in such a way that he is able to search the abdominal cavity for the presence of abnormal conditions or he may display a masterly inactivity and wait for a definite indication of the necessity for and of the extent of surgical measures.

FISTULA-IN-ANO.

MEDICAL students of the last generation were taught that the association of *fistula-in-ano* with some form of tuberculosis, generally pulmonary, was not uncommon. The fistulæ were regarded as tuberculous. At the same time there was a wide divergence of opinion in regard to the matter.

Attention has recently been drawn to this difference of opinion in a study by Dr. B. R. Clarke.[1] Dr. Clarke points out that according to Melchior 61% of fistulæ are tuberculous, while Gordon Watson, writing in Gask and Wilson's "Surgery," places the percentage no higher than three. He compares these figures with those published by such writers as Lockhart-Mummery, Powell and Hartley, Fishberg, Rickmann and Walsham. The percentage of fistulæ amongst tuberculous patients reported by these authors was in every instance lower than four. The lowest figures are those of Walsham who found five fistulæ and two ischio-rectal abscesses among eight hundred and ninety-one persons suffering from pulmonary tuberculosis. Dr. Clarke points out that the diagnosis was not always confirmed by these writers by pathological examination. In order that a *fistula-in-ano* be accepted as tuberculous it is necessary to find tubercle bacilli in the discharges or in sections of the tissue or to produce tuberculous lesions as the result of animal experimentation. Dr. Clarke has found a record of *fistula-in-ano* in ninety among 6,465 patients suffering from pulmonary tuberculosis, eighty-one were males and nine were females. During 1924 one hundred and eighty-five patients with pulmonary tuberculosis were admitted and eleven of them (6%) suffered from fistula or ischio-rectal abscess. He found that *fistula-in-ano* is thirteen times commoner in male tuberculous than in non-tuberculous patients. Dr. Clarke collected one hundred and nine cases of fistula associated with pulmonary tuberculosis. In sixty-one cases the fistula preceded the diagnosis of the lung condition, in five cases the records did not reveal which condition was diagnosed first and in forty-three cases the pulmonary tuberculosis was discovered first. Twenty-nine patients were operated on before the tuberculous condition was recognized. In seventeen the pulmonary tuberculosis appeared within three years of the operation and in ten the fistula was apparently healed. Dr. Clarke does not explain whether the discharges were examined in the one hundred and nine cases or whether any other steps were taken to ascertain the tuberculous nature of the fistula. A non-tuberculous fistula may occur in a tuberculous patient. He concludes with the practical suggestion that pulmonary tuberculosis should be rigorously excluded in every case of fistula before surgical treatment is considered.

[1] *Tubercle*, March, 1926.

Abstracts from Current Medical Literature.

PHYSIOLOGY.

Effects of Anoxæmia on Heart and Circulation.

JANE SANDS AND A. C. DE GRAFF (*American Journal of Physiology*, October, 1925) have made a critical study of the effects of anoxæmia on the circulation, using dogs under chloretone and morphine analgesia. They find that during the progressive increase in the degree of anoxæmia previous to the circulatory failure at the crisis which generally occurs when the oxygen concentration has been reduced to about 9%, there are definite indications that the circulation improves and thus helps to supply the tissues with normal volumes of oxygen. When the vagi are intact and vagal tone is good, the heart accelerates progressively, partly on account of diminution of vagal tone, but to a lesser degree from an accelerator stimulation or a direct cardiac action. The systolic pressure tends to increase and the diastolic pressure either remains unaltered or slightly increases, thus tending to increase the pulse pressure. This may be due to a combination of cardiac acceleration and reduced peripheral resistance, but as anoxæmia becomes even moderately severe, it is certainly due to augmented systolic discharge. Anoxæmia reduces the effective venous pressure and also raises the initial tension in the left ventricle and increases the velocity of ventricular contraction. During the course of progressive anoxæmia these two factors remain evenly balanced and tend to maintain a normal systolic discharge, but as the demand for oxygen becomes more intense, the latter factor always predominates and increases the systolic discharge. The circulatory crisis in anoxæmia occurs when the oxygen supply is diminished to a point at which the hitherto beneficial influence on the heart rapidly changes to a deleterious action. The first evidences of such failure are found in the decline of systolic and diastolic pressures and a reduction of pulse pressure. This cardiac failure definitely precedes the final slowing of the heart. Evidence is incomplete as to the exact mechanisms affected by oxygen lack. Up to the crisis it is unnecessary to assume any direct effect of anoxæmia on the heart, for all the stimulating effect can be accounted for by its effect on the vagus and accelerator mechanisms. The final failure is no doubt due to a direct effect of oxygen deficiency on the heart.

Humoral Stimulation of Gastric Secretion.

ALTHOUGH a humoral mechanism for gastric secretion has been postulated for many years, evidence clearly proving such a mechanism has not been offered. Blood transfusion and cross circulation experiments have given very inconclusive results. A. C. Ivy and J. I. Farrell (*American Journal of Physiology*, November, 1925) have endeavoured to obtain evidence by a new procedure, namely the subcutaneous transplantation of a gastric pouch. A gastric pouch was transplanted into the mammary glands of five female dogs. The transplanted pouch manifests a continuous secretion which never contains free acid and only a small amount of combined acid. Such a pouch secretes a gastric juice containing acid after a meal and after histamine and gastrin administered subcutaneously. The latent period of response after a meal is from two to six hours. Mechanical distension of the pouch also stimulates the glands. The evidence presented shows that a humoral mechanism is one of the mechanisms concerned in the genesis of gastric secretion. It does not, however, prove or disprove a hormone hypothesis. It might well be due to secretagogues present in or resulting from the digestion of the food.

Extrahepatic Formation of Bilirubin.

IN work previously reported data were presented proving that bilirubin is formed in the body even after the removal of all the intraabdominal organs, thus conclusively demonstrating that there is an extraabdominal source of bilirubin and that neither the liver nor any other intraabdominal organ is necessary for its formation. F. C. Mann, C. Sheard, J. L. Bollman and E. J. Baldes (*American Journal of Physiology*, November, 1925) have attempted to locate the site of the formation of bilirubin after removal of the liver. By spectrophotometric methods they examined the venous blood issuing from various organs and the arterial blood entering these organs. In most of the vascular areas the bilirubin content of specimens of blood from the two systems was the same, but the bilirubin content of the venous blood was greater than that of the arterial blood of spleen and bone marrow. This shows that bilirubin is formed in the spleen and bone marrow.

Effect of Radiation on Metabolism.

WHILE it is known that radiant energy exerts a strikingly curative effect on rickets and other conditions of malnutrition, little work seems to have been done on the action of radiant energy on the metabolism of normal animals. H. S. Mayerson, L. Günther and H. Laurens (*American Journal of Physiology*, January, 1926) have studied the effect of carbon arc radiation on metabolism in the dog. As a source of radiant energy they used a "Majestic" arc which gives a spectrum very like that of sunlight. At forty centimetres distance the arc operated at twenty-five ampéres, emits energy with an intensity of $1{\cdot}5415 \times 10^{-3}$ gramme calories per square metre per second. This is comparable to the average seasonal solar radiation intensity in Washington for three hours at noonday. The spectral distribution of the energy is, however, quite different from sunlight, approximately 50% being ultraviolet, 11% visible and 39% infrared, as contrasted with solar radiation in which not more that 1% to 2% is ultraviolet, 40% to 50% visible and 50% to 60% infrared. Irradiation with this lamp increases the endogenous nitrogen metabolism, stimulates the absorption of calcium and phosphorus from the intestine and usually decreases the amount of blood sugar. Serum phosphorus is definitely increased by irradiation, in some cases as much as 300%. On moderate exposure to arc lamp radiation there is a balance between serum calcium and phosphorus. Excessive or repeated irradiation also increases serum calcium, the rise coming simultaneously with that of phosphorus.

Rest and Basal Metabolism.

THE term "basal metabolism" as currently accepted, implies the minimum of energy necessary to keep the human machine functioning while in repose, awake and twelve hours after the last meal. Thirty minutes of complete muscular repose are commonly considered as meeting the requirements. Recently it has been suggested that the minimum metabolism may not be secured until after a considerable period of rest in bed. This is an important point to determine, for it is necessary to know whether a possibly much needed hospital bed must be sacrificed for a night to insure basal conditions. F. G. Benedict and E. E. Crofts (*American Journal of Physiology*, October, 1925) have put this question to the test in a series of carefully planned experiments. They find that the basal metabolism measured after a night's sojourn in bed with the body well covered, warm and relaxed, is but insignificantly increased in a period following the muscular exercise of rising, bathing, walking in wintry weather for ten minutes and climbing three flights of stairs, provided that after such exercise the subject lies clothed and lightly covered in a room at about 20° C. for a period of thirty minutes.

Tonus After Sympathectomy.

THE failure of certain workers to show a loss of plastic tone in muscles after sympathectomy and decerebration has been attributed by Royle to two factors, the selection of an unsuitable animal and neglect to wait a long enough time after removal of the sympathetics before making the test. W. J. Meek and A. S. Crawford (*American Journal of Physiology*, October, 1925) have used dogs on which few experiments have been made, and have allowed considerable time to intervene in order to meet the last criticism. The dogs were kept for thirty-three to seventy-seven days after sympathectomy and then decerebrated. Before decerebration the un-

anæsthetized dog was placed on its back and tested for knee jerk in each hind limb. In no case was there in either leg the phenomenon of the "shortening reaction" which Sherrington's work indicates as characteristic of plastic tonus. After decerebration both the normal legs and legs whose sympathetic nerve supply had been destroyed, would stay in position in any degree of contraction or extension. The conclusion is drawn that in the dog plastic tonus does not depend on sympathetic innervation.

BIOLOGICAL CHEMISTRY.

Estimation of Fat in Blood.

C. P. STEWART AND A. C. WHITE (*Biochemical Journal*, August, 1925) have devised an accurate method for estimating fat in blood. The small amount of fatty acid obtainable from two cubic centimetres of blood has rendered it impossible to utilize with any degree of accuracy the hydrolytic method of estimation in which the fat is saponified by sodium hydroxide and the fat excess alkali titrated with acid. The new microburette, however, makes this possible. The authors find that it is possible to carry out this operation without trouble, provided that a few simple precautions are observed. It is obvious that for such estimations both reagents and apparatus must be accurately standardized. The results have been compared with those obtained with pure solutions of tripalmatin and with figures for blood to which known amounts of tripalmatin have been added.

Cerebro-Spinal Fuid.

IRVINE MCQUARRIE AND A. T. SHOHL (*Journal of Biological Chemistry*, December, 1925) have studied the acidity of cerebro-spinal fluid. Many attempts had been made previously to measure the hydrogen ion concentration of spinal fluid, some by colorimetric and others by electrometric methods. Foa in 1906 found pH = 7·2, but later observers obtained values as 10, greater than 9, 8·1, as well as 7·2 and 7·3. No attention can be paid to much of this work whose authors took no cognizance of the importance of tension of carbon dioxide. Loss of carbon dioxide led investigators to obtain a value for pH much too low. A review of the literature shows that those who used most precautions against any escape of carbon dioixide, obtained figures similar to those found for blood, while those who neglected the factor, got results between 9·0 and 10. It is therefore of importance to prevent any loss of carbon dioxide. An apparatus has been used by the authors in which the spinal fluid never comes into contact with air, is collected over mercury and from which no transference takes place. The spinal fluid is collected and determined in this apparatus. The colorimetric comparison has been made at the temperature of the body. The temperature is of importance for two reasons. The phosphate solutions used as buffers become more alkaline when heated. As a result a standard containing phenol red at pH = 7·20 becomes more red on being heated from 20° C. to 38° C., so that at 38° C. it matches a standard of 7·28 at 20° C. In the second place the indicator which is a weak acid, becomes stronger and binds more base. More of it thus appears in the alkaline or red form. The authors have used the colorimetric method of Hastings and Sendroy and made their comparisons at 38° C. They have made comparisons of the hydrogen ion concentrations of blood and of spinal fluid determined simultaneously on the same individual. The results obtained show that the figures for blood and for spinal fluid are the same within the sum of the errors of the two determinations. As the samples have been taken from persons suffering from different infectious diseases, from healthy persons and from dogs, the range of hydrogen ion concentration for the blood has varied from pH = 7·17 to pH = 7·47.

Antirhachitic Vitamin in Eggs.

J. S. HUGHES, L. F. PAYNE, R. W. TITUS AND J. M. MOORE (*Journal of Biological Chemistry*, December, 1925) have studied the relation between the amount of ultraviolet light received by hens and the amount of antirhachitic vitamin in the eggs produced. This study has been undertaken to examine the validity of the belief that variation in the hatchability of the eggs produced by hens receiving different amounts of ultraviolet light is due to a variation in the content of antirhachitic vitamin in the egg. Four groups of chickens have been investigated. In two groups sunlight was permitted and in two groups all sunlight was filtered through glass. Two groups were exposed for thirty minutes daily to the irradiation of a mercury vapour lamp. The results have shown that the amount of ultraviolet light which a hen receives, is an important factor in determining the antirhachitic vitamin content of the eggs which the hen produces on a diet poor in vitamin *D*. It has further been noted that eggs poor in antirhachitic vitamin have a lower percentage of hatchability than eggs with a high content of antirhachitic vitamin.

Irradiated Milk.

H. STEENBOCK, E. B. HART, C. A. HOPPERT AND A. BLACK (*Journal of Biological Chemistry*, December, 1925) have studied the influence of direct irradiation of milk with a quartz mercury vapour lamp. They have found that its antirhachitic potency is materially increased. Irradiation has been carried out by exposing the milk in an iron pan at a distance of sixty centimetres (two feet) to light from a burner of voltage forty to sixty and four ampéres. The time of exposure has been thirty minutes, the milk being tipped from side to side during the exposure. This milk has been fed to rats of about sixty grammes weight fed with a ration of yellow corn, wheaten gluten, calcium carbonate and sodium chloride. This ration always produces rickets of uniform severity in rats in a few weeks. After twenty days when moderate rickets had developed the diets have been supplemented with milk for ten days. The quantity of milk has been carefully measured. After ten days the rats have been killed and the distal ends of the radii and ulnæ split and stained with silver nitrate. In an experiment with cow's milk the antirhachitic potency of the irradiated milk has been roughly ten times that of ordinary milk. In another experiment goat's milk has been used, as it has been desired to test also the effect of irradiation of the animal and the size of the goat is more suitable. Direct irradiation of the milk increased its antirhachitic potency over twenty times. Irradiation of the animal has not been so effective, the increased potency of the milk being about one quarter of its potency when irradiated directly. The authors suggest that every producer of high grade milk should irradiate his cows artificially as irradiation by sunlight is so variable.

Metabolism of Rats Deprived of Vitamins.

A. F. MORGAN AND D. F. OSBORN (*Journal of Biological Chemistry*, December, 1925) have studied the partition of urinary nitrogen as urea, ammonia, allantoin, uric acid and creatinine in rats fed on a diet adequate in all respects except as to vitamin *A*. As controls they have used three young rats fed on a diet adequate in all respects. They have recovered between 45% and 64% of the total nitrogen in the urine. The relative amounts of allantoin in the urine decreased with gain in body weight and increased with loss in body weight. This corresponds to 0·144 gramme of allantoin nitrogen per kilogram body weight correlated with a gain of 28·8 grammes weight of body. They have fed nine young rats on the ration poor in vitamin *A*. The percentage of recovery of total nitrogen ranged between forty-six and fifty-nine. The relative amounts of allantoin lessened with loss in body weight. The average amount of allantoin was 0·165 gramme allantoin nitrogen per kilogram body weight correlated with an average loss of 32·8 grammes body weight. The authors have suggested that in the absence of vitamin *A* the animal organism fails to produce purin containing substances from the usual sources possibly arginine and histidine, but continues to utilize again and again portions of substances containing the purin ring which are ordinarily excreted in the form of allantoin.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Lister Hall, Hindmarsh Square, Adelaide, on February 25, 1926, DR. C. T. C. DE CRESPIGNY, D.S.O., the President, in the chair.

Fractures.

DR. P. J. S. CHERRY read a paper entitled "Treatment of Fractures in a Casualty Hospital" (see page 481).

DR. BRONTE SMEATON read a paper entitled "Notes on Fractures and Dislocations of the Upper Extremity" (see page 487).

DR. MALCOLM L. SCOTT read a paper entitled "Fractures of the Lower Extremity" (see page 485).

Adiposis Dolorosa.

DR. F. S. HONE read the notes of two cases of *adiposis dolorosa* occurring in two sisters (see page 488).

Gunshot Wound of the Jaw.

DR. H. S. NEWLAND, C.B.E., D.S.O., showed a boy, aged fourteen years, who had suffered from an extensive injury to the lower jaw fourteen months previously as a result of a gunshot wound. The chin and mandible had been destroyed as far as the second molar tooth on either side. The lower lip was absent, the chin collapsed and almost on a level with the neck, the skin had been retracted as far back as the anterior part of the root of the tongue. The tongue had been uninjured and hung outside.

The first procedure in the treatment a few minutes after the accident had been to apply a Gunning splint to the remaining molar teeth to keep the ends of the mandible in position. For six months an endeavour had been made to build up the patient's general condition. On December 11, 1925, a lower lip had been modelled from flaps from either cheek together with some skin from the neck. This had healed at once and on January 3, 1926, a hooded flap had been marked out on the forehead with a base above each ear, this had been undercut and tubularized. On February 10, 1926, by a transverse incision across the neck and by means of undercutting a bed had been prepared for the tubular forehead graft which had been swung down across the face into position and stitched to form a chin. The forehead wound had been grafted by a whole thickness skin graft from the chest. The present state of the patient was thus described. It was intended later on to sever the pedicles of the graft in the neck and swing them back to spaces which had been left for them in the temporal regions. A bone graft would also be inserted into the reconstructed chin from the crest of the ilium.

Volkmann's Paralysis.

DR. H. GILBERT showed a case of Volkmann's ischæmic paralysis following a fracture of the forearm.

Radiography of the Gall Bladder.

DR. H. C. NOTT exhibited a series of radiograms of the gall bladder taken after the administration of sodium tetraiodophenophthalein.

One patient had had an intravenous injection of the salt and two had been given pills by the mouth.

In each case the gall bladder was very well outlined and appeared normal according to the present ideas on the subject.

Dr. Nott pointed out that the patients had all complained of obscure abdominal symptoms which could have originated in the gall bladder and by this method of examination he was able apparently to exclude the gall bladder as the seat of the trouble.

Public Health.

ANTERIOR POLIOMYELITIS IN NEW ZEALAND.

THE following report dealing with the recent epidemic of anterior poliomyelitis in New Zealand has been received by us in response to a request made to the Director-General of Health of the Dominion. In view of the importance of the subject and the interesting nature of the report, it is published in full.

REPORT ON THE INFANTILE PARALYSIS EPIDEMIC IN NEW ZEALAND, NOVEMBER, 1924, TO MAY, 1925.

The epidemic began on November 25, 1924, near Wellington, North Island, and quickly spread to Wellington. It gradually invaded the country towns of the North Island in characteristic irregular manner and later Auckland City in the north.

The South Island was not affected until late in January and then the incidence was definitely lower. About four months was the greatest duration of the epidemic in any district.

In the 1916 epidemic also when 123 deaths occurred, incidence and death rate were much greater in the North Island, namely from north to south. This is seen in the following table:

District.	Notified Cases.	Deaths.
Health District of Auckland	546	66
" " " Wellington ..	382	46
" " " Canterbury ..	65	8
" " " Otago	25	3
	1,018	123

New Zealand's latitude ranges from 34° to 47° comprising as it does two long islands and the climate ranges from subtropical in the north to temperate in the south.

TABLE SHOWING NOTIFICATIONS AND DEATHS FROM INFANTILE PARALYSIS IN NEW ZEALAND ANNUALLY.

Year	Notifications.	Deaths.
1912		2
1913		2
1914		9
1915	10	3
1916	1,018	123
1917	54	10
1918	6	4
1919	11	1
1920	76	2
1921	267	9
1922	98	9
1923	17	2
1924-25	1,257	166

Eight years, therefore, have passed since the last epidemic.

The 1924-25 epidemic began on November 25, 1924, at Petone and quickly spread to Wellington. Its incidence until and including April 30, 1925, was as follows:

Whole Dominion: Population 1,300,000, 1,257 cases notified, 166 deaths.

Auckland Health District: Population 398,000, 310 cases notified, 61 deaths.

Wellington Health District: Population 476,000, 604 cases notified, 70 deaths.

Canterbury-Westland Health District: Population 250,000, 277 cases notified, 27 deaths.

Otago-Southland Health District: Population 207,000, 66 cases notified, 8 deaths.

Expressed as a ratio per 100,000 of mean population this produces the figures seen in the following table:

District.	Cases Notified.	Deaths (per 100,000 of Population).
Whole Dominion	97	12·7
Auckland Health District	78	15·3
Wellington Health District	127	14·7
Canterbury-Westland Health District	111	10·8
Otago-Southland Health District ..	32	3·9

Severity.

The most severe epidemic of infantile paralysis recorded to date was that of 1916 in the north-eastern States of America.

The population of these north-eastern States in 1916 was roughly thirty-two millions and in that year there were 72 cases per 100,000 of population and 16 deaths per 100,000 of population.

In this recent epidemic in New Zealand there were 97 cases per 100,000 of population and 12·7 deaths per 100,000 of population.

To judge from the death rate then which is the most reliable index of severity, the epidemic we have lately experienced, can be regarded as comparatively severe.

This year in New Zealand the number (97) of cases notified per 100,000 of population is high. This probably spells careful diagnosis and free notification of mild cases which is an aid to prevention of spread and is a satisfactory feature.

In the early stages of the epidemic a committee set up by the Council of the British Medical Association and the Department of Health promulgated to all medical practitioners in the Dominion a pamphlet summarizing the symptomatology of this disease and in order to lessen the spread of this disease emphasizing the importance of notifying all patients capable of conveying infection.

Age incidence of cases notified was as follows: 95·7% of patients were under twenty years of age. 92·1% of patients were under fifteen years of age. 61·9% of patients were under six years of age.

The optimum ages were: Children of two years and under three contributed 15% of the total. Children of three years and under four contributed 13·7% of the total.

These ages give the highest attack rate.

On either side of these children of one year and under two contributed 11·9% of the total; children of four years and under five contributed 10% of the total. This age incidence may be called typical.

Hospitalization of Patients.

The reports we received from the medical attendants concerning the early cases indicated severity of type and it was feared the paralysis result throughout the Dominion would be formidable. Although, however, 166 deaths have occurred, later reports from nearly all centres state that recoveries from paralysis have been unexpectedly numerous.

Our death rate was comparatively high and naturally one would have expected a high paralysis rate also. That appears to be the usual concomitant of severe epidemics of this disease. Either, then, an anomaly is presented by our high death rate and low paralysis rate or, what is more probable, a distinct advance has been made in treatment. New Zealand is well supplied with hospitals. The great majority of the patients were promptly sent to hospital. Careful splinting and posturing have been the usual practice and in the larger centres this has been done under the direction of surgeons who have received special tuition in orthopædic work. Convalescent serum has been used in considerable measure and quite half the medical attendants claim that, if given in the pre-paralytic stage, it is curative. This claim, however, must meantime be considered with some reservation owing to the comparatively small population dealt with.

Prevention of Spread.

I may say the loyal manner in which the general public have observed the precautionary measures deemed expedient and the complete cooperation of the great majority of medical practitioners with the officers of the Department of Health have caused the latter considerable gratification.

Within our memories infantile paralysis was considered not to be an infectious disease. It has all along been far more prevalent than we realized. To judge from observations in many epidemics the mild systemic cases must in epidemic periods be very prevalent indeed and even in normal years, when from two to ten deaths are recorded in the Dominion, there are probably far more mild systemic cases in particular localities than are recorded.

Many experienced observers believe that the disease in mild form is very common indeed, that in epidemic years immunity is acquired by a large proportion of the child population and in non-epidemic years by means of a less virulent strain of virus there is a far greater degree of immunity thus conferred than we have hitherto realized. It is thought that this inestimable immunity factor is the important element underlying the mystery which surrounds infantile paralysis and its means of spread.

In common with other infectious diseases infantile paralysis is probably most conveyable in its acute stage. Its incidence preponderates enormously in persons under sixteen years of age. Mild systemic cases are believed to be very much more common than frank meningitic or paralytic cases. A great number of them probably are not recognized as such and there is every chance that medical opinion or aid will not be sought. Hence, in addition to the usual measures of notification, hospitalization of the patient and isolation of susceptible contacts, it was decided definitely to restrict the congregation of children in areas where infection was believed to be rife.

As with diphtheria there is apparently an age immunity as well as an acquired immunity. It is an infantile disease and we can reasonably claim that a great majority of those susceptible children of tender years who have escaped this epidemic, will also escape a future visitation of infantile paralysis several years hence.

It was decided not to isolate adult contacts when a child was sent to hospital, unless of course such adults had pyrexia or some symptoms attributable to this disease or unless their occupation meant close contact with numbers of children. While it is admitted that an adult may become a carrier, probably this is a rare event and in any case there is no practicable means of testing such a condition or of knowing how long it may persist. The isolation of adults has a damaging economic effect and there is as yet insufficient evidence to justify our isolating all adult contacts when the patient has been sent to hospital in view of the fact that apparently very few actually contract the disease.

Clinical Features.

(A Review of the Cases of Polioencephalitis in the Wellington Hospital, by W. S. Robertson, M.B., Ch.B., Wellington Hospital.)

Incidence.

The age incidence, as in other epidemics, is from two to five years. The youngest patient was three weeks, the oldest forty-six years. There has been no gross sex preponderance. The incubation period in some group cases was about five days. A great similarity of type was shown in the early cases, the fair, flaxen-haired, blue-eyed children being predominant. The weedy child, the guttersnipe and the ragamuffin were conspicuous by their absence; all the patients with few exceptions being plump, well-fed, perfect specimens of childhood.

The numerical incidence of admissions for the town district is about 8‰ children below the age of ten years. There appears to be no doubt that the whole community is exposed to infection, but that owing to a natural or acquired immunity the large majority of children escape or attack the infection so successfully that the symptoms of invasion never become severe enough to cause any anxiety to the parents. It is equally certain that these mild and abortive cases are instrumental in spreading, the infection by infective nasal or bowel discharges.

A prominent feature of the epidemic has been the fact that for each child of a family admitted there has been a history of transient sickness among other members of the family at some time. Lumbar puncture was done on some of these contacts and in three cases an increase of the cell count in the spinal fluid was reported.

Symptoms.

The cases have all formed a very similar clinical picture. The onset of symptoms on the whole has been fairly rapid, the prodromata consisting of lassitude, anorexia, constipation and headache, the whole culminating in a sharp attack of vomiting with maybe a mild pyrexia of 99° to 100° F..

The headache is not localized at this stage. The vomiting, usually one attack only, has been a very consistent early symptom. A large percentage gave the following history of onset: The child went to the beach, played in the sun all day, complained of not feeling well in the afternoon, intense headache and vomiting that night and so on. A somewhat similar history was obtained from some of the adult sufferers in country districts, that is the patient had been out working in the hot sun all day—intense headache and vomiting occurred at night with suspicion of "sunstroke." From these cases it would appear that the sun's rays have at least a predisposing effect on some patients. This stage lasts some two or three days, the symptoms becoming more severe, the headache predominating and usually becoming localized to the occipital region. From a state of lassitude the child gradually drifts into a state of extreme irritability and dislike of being touched or disturbed. Constipation is still in evidence and the pyrexia may be 38·3° to 38·9° C. (101° to 102° F.). At this stage there appears to be a general hyperæsthesia, the tenderness being in some cases so acute as to make the child scream when touched.

Sore throats, coryza, nose bleeding and enlargement of cervical lymph glands have occurred rarely. Irrespective of the pyrexia there is excessive general sweating and the face has a typical cyanotic dusky flush with a definite circumoral pallor, this flush lasting for some days after subsidence of acute symptoms.

Two patients, one of whom died, manifested a diffuse general dusky red macular rash. Several patients had a discrete papular rash on the trunk, probably miliarial in origin. Flea bites were unknown.

Condition on Examination.

Clinical examination three to four days from onset of illness reveals the following: Extreme irritability, in some cases drowsiness or coma; tenderness of limbs, back and neck; rigidity of cervical spine, with maybe slight head retraction; pain on attempting to bend the head; photophobia; occasional twitching of limbs; possibly some slight weakness of a limb movement or inability to stand; excessive sweating.

Reflexes.

The abdominal reflexes and knee jerks may be increased or diminished, equal or unequal. The plantar responses are more often extensor in type. Kernig elicitation causes a definite pain in the popliteal space, but little hamstring spasm. No anæsthesia changes have been observed. Urinary incontinence is common.

Cerebro-Spinal Fluid.

The spinal fluid is under increased pressure, with increase in the cell count ranging from ten lymphocytes per cubic millimetre to 2,700 per cubic millimetre. There is increase of globulin content. There is a definite opalescence of the fluid plainly visible in daylight. These changes appear to return rapidly to normal with appearance of paralysis.

Blood-Count.

Differential blood-counts were done in all cases and proved too variable to be of the slightest material use.

Nasal and Throat Swabs.

Nasal and throat swabs were taken in several cases, but the virus could not be isolated.

Course of Illness.

At this stage of the disease two courses are open: (i.) The symptoms gradually subside, the irritability being the first one to disappear. This is the true, abortive type and represents the stage beyond which the disease does not usually develope when serum is administered early or (ii.) the various types or syndromes appear as to the particular localization or extension of the pathological process in the central nervous system.

Dromedary Course.

Many patients gave a history of having run the "dromedary course" of the disease, having suffered from the earlier prodromal symptoms for two or three days and then, being apparently quite well, they had been allowed up. After a variable quiescent period of one or two days these patients had then been suddenly struck down by a gross exaggeration of typical symptoms with extensive rapid paralysis or death. The two stages definitely mark: (i.) The period of infection and (ii.) the period of invasion.

Classical Types.

The classical types met with are as follows, most of them passing through a stage of meningitis with more or less severe meningeal signs: (i.) Meningitic, (ii.) spinal, (iii.) bulbar, (iv.) encephalitic, (v.) facial, (vi.) Landry, (vii.) neuritic, (viii.) cerebellar.

Meningitic Type.—The meningitic type is a common type and is an exaggeration of symptoms of the abortive type, that is there are definite cervical rigidity, head retraction, maybe opisthotonos, excessive tenderness of limbs, intense headache and irritability, positive Kernig and extensor plantar responses. Some muscular weakness or paresis is always evident in this type, but it is not extensive and tends to clear up quickly. Strabismus is a sequela of this class.

Spinal Type.—The spinal type may have comparatively mild prodromata, the first suspicion of any illness in the child being very often inability to stand or to raise a limb. Careful inquiry will usually elicit a history of the child having been out of sorts or fretful. The paralysis here is usually extensive and complete often bilateral. The younger children are mostly of this class.

Bulbar Type.—The bulbar type has a high mortality rate, children of eight to twelve being specially liable. The onset and initial course of the disease is acute, the patient rapidly becoming acutely ill, passing into a state of drowsiness, dyspnœa and inability to swallow. The intercostal and extrarespiratory muscles become paralysed, breathing is diaphragmatic, coma supervenes and the case usually terminates fatally, *post mortem* examination revealing definite macroscopical hæmorrhagic areas on the floor of the fourth ventricle. In one case only of this type did recovery occur.

Encephalitic Type.—In the encephalitic type the irritability, headache and drowsiness are the chief features. There are spasticity of limbs extensor plantar responses, increased reflexes *et cetera*. The condition tends to clear up completely with no mental impairment. One patient had loss of speech for three weeks with sudden complete recovery.

Facial Type.—The facial type is common and may come on with little in way of prodromata. Generally the paralysis is unilateral, incomplete and clears up very slowly and incompletely.

Landry Type.—A few patients appeared to be rapidly overwhelmed with a general weakness, paresis, paralysis involving all limbs, coming on in course of twelve to twenty-four hours and ending fatally. No definite "ascending" sequence of paralysis could be followed.

Neuritic Type.—The neuritic type is characterized by a more acute and more persistent limb pain and tenderness, followed by weakness and paralysis coming on over a relatively long period of three or four days. The early symptoms are mild.

Cerebellar Type.—Two patients only showed a semblance to the cerebellar type, with choreic or ataxic movements of arms. Both recovered with no further sequela.

Paralysis.

Paralysis comes on three to four days from onset of symptoms, goes through a definite sequence of weakness, paresis, paralysis over a variable period of twenty-four to forty-eight hours, becoming much more extensive than the actual residual paralysis and maybe clearing up in two to five weeks, the part first affected being the last to recover.

The deltoid, triceps, quadriceps, ankle dorsiflexors and facial muscles are the muscles most affected. No muscle or muscle group appears to be immune.

Diagnosis.

Lumbar puncture has proved of immense value in diagnosis in the preparalytic stages. Other important early diagnostic signs are cervical rigidity, presence of Kernig's sign, extensor plantar responses, deep muscular tenderness, muscular weakness, especially unilateral.

Suspect conditions included, sapræmia, brain abscess, lumbago, pyomeningitis, tuberculosis, meningitis, rheumatism, osteomyelitis, appendicitis.

Treatment.

The routine treatment carried out was:

(i.) Recumbency, with light diet and aperient.

(ii.) Lumbar puncture, 1% "Novocaine" intradermally being used. For diagnostic purposes five cubic centimetres of fluid were removed, but if serum were indicated, fifteen to twenty cubic centimetres were removed very slowly and ten to fifteen cubic centimetres of convalescent serum at body heat introduced intrathecally, also very slowly by gravity.

(iii.) Serum administration. The blood serum of patients who had been convalescent for from two weeks to three months, was used extensively in all preparalytic cases with apparent gratifying effects, one favourable case only not reacting. End results will be published later. In some of the older cases twenty cubic centimetres of serum were given intravenously in addition. If necessary the dosage was repeated in twenty-four hours. No bad effects were experienced either from the lumbar puncture or from the administration of serum. The serum was prepared by titration and inactivation according to the technique of "Nelson's Loose-leaf Medicine."

Following serum administration the lower end of the patient's bed was raised on blocks for twenty-four hours.

(iv.) Immediate splinting irrespective of the presence of weakness or paralysis by light, close-fitting supports was carried out in all cases.

Plaster spinal beds were made for all patients under three or four years of age or for any patient showing spinal or abdominal weakness. Plaster bed boots above knees with feet at right angles were made for older patients, who were nursed on firm mattresses with fracture boards, the limbs being steadied by sandbags and protected from pressure by cradles.

The arms were supported at 90° shoulder abduction, elbows were semi-flexed and forearms were semi-prone. Immediate splinting limits the extent of an oncoming paresis, hastens the recovery of a paretic muscle and prevents deformity. The whole plaster bed gives a supported relaxation of the whole of the body in true physiological position and, being supported on two boxes at either end or on a gas-pipe framework with a bed pan beneath, the disturbance of the child is minimal. Urotropine was used in early cases of the epidemic only and appeared to be of no benefit. For the bulbar type, adrenalin injected subcutaneously or three cubic centimetres of one in a thousand in two cubic centimetres of saline solution given intrathecally proved of some value. Nasal feeding and oxygen inhalation were utilized.

Frequent lumbar puncture is indicated for the meningitic type, giving great relief to the headache and restlessness.

Convalescence.

Tentative reeducative measures are commenced at expiry of the acute stage and gradually developed as the muscles recover. Massage and electricity have not been used in any cases of under three months' duration and then in selected cases only. Toy balloons are being found of great value in reeducating the younger children by getting them to attempt to touch an object which naturally attracts their eye. Patients before being allowed to stand are got into a sitting-up position by graduated rises on a bed rest. When ready for walking the patient wears calipers, long or short, abduction frames and other ambulatory supports to protect weakened or paralytic muscles.

After three years operative measures are indicated for a non-improving patient, muscle transplants *et cetera* being done to improve stability.

Prognosis.

Electrical reactions are of no real value in this condition. Undoubtedly there are cases which are abortive without treatment, but just as certainly serum, if administered in time, will abort an attack which would otherwise become serious. Unfortunately in the early stage of the epidemic the majority of the cases were diagnosed by the appearance of the paralysis and the patients were then on admission in too late a stage to receive benefit from serum treatment.

Later on, however, the cases were being recognized by the general practitioners in a much more favourable stage for serum administration. The prognosis for the child of ten to twelve as regards life is not good, the bulbar type predominating at that age. As regards muscle recovery, no case is hopeless under twelve months.

Conclusion.

To deal successfully with these cases, early diagnosis is essential and lumbar puncture gives ready means to accomplish this. Serum to be of any use must be given in the preparalytic stage and is then almost specific. Immediate splinting has proved its usefulness.

Pathological and Bacteriological Investigation.

(Memorandum for the Director-General of Health, Wellington.)

DEAR SIR: I respectfully beg to present my report on work done for the Department during the recent epidemic of poliomyelitis. The work was commenced at the request of the Department on January 19 and was continued throughout the rest of this month and the greater part of February and March. The Department allowed me the assistance of Mr. A. Pierard who was kindly released by Mr. Hurley, the Government Bacteriologist and Mr. de Clive Lowe, a final year medical student from Dunedin. At the time I commenced the work, the epidemic was in full swing in Wellington and I had already examined a number of cases *post mortem*, but beyond the histological examination of these nothing further was done. I decided to investigate what material was available from the epidemic:

(i.) From the pathological standpoint, to determine the nature and extent of the lesions as compared with similar epidemics elsewhere.

(ii.) From a bacteriological point of view in order, if possible, to identify and culture the causal organism.

(iii.) By animal inoculation to produce experimental poliomyelitis with a view to carrying out immunological work later on. In this latter course I was advised by Professor Hercus with whom I discussed the question.

As regards our present knowledge of the disease, there are two distinct schools of opinions as regards the ætiological factor—that of Flexner and Noguchi who attribute it to a virus which has the form of minute globoid bodies which grow under anærobic conditions and which are capable of passing through the finest filters, and that of Rosenow and his followers who say that the causal organism is a tiny pleomorphic streptococcus which attains its minimum size under anærobic conditions and will then pass through a Berkefeld filter.

We have studied the available literature on the subject (mainly in the form of abstracts) and the consensus of opinion seems to favour the ætiological significance of the globoid bodies. Rosenow's view obtains but little support and the majority of workers have been unable to reproduce his findings. Support is given to the coccus theory by Nuzzum and Willy who in 1917 during an epidemic of poliomyelitis used the serum of horses immunized against this particular streptococcus, with apparent success and also by Max Herzog who inoculated monkeys, lambs and rabbits with this streptococcus and said they produced the characteristic clinical and pathological picture of poliomyelitis. Amoss reports how an American worker, named Bull, failed to reproduce Rosenow's findings and concluded that in animals inoculated with the streptococcus the focal lesions which were present, were not identical with the lesions of poliomyelitis. He also failed to immunize monkeys. W. G. Smillie, of the Rockefeller Institute, could not prove the implication of the streptococcus in the

disease and looked upon it as a *post mortem* or secondary invader. Ludwig Hektoen found that the streptococcus would not pass the finest filters, whereas the virus would and found in many of the rabbits inoculated with the streptococcus the ordinary lesions of a streptococcus septicæmia. Amoss and Eberson failed to produce poliomyelitis after inoculating a series of animals with Rosenow's streptococcus. Many workers including Hektoen, George Mathers and Leila Jackson have found the streptococcus fairly constantly present in the tissues from persons suffering from the disease, but do not consider that it plays any part in the ætiology. Flexner and Noguchi have recovered and cultured the globoid bodies from persons suffering from the disease, have inoculated monkeys with it and again recovered it. Further, after a series of cultures and subcultures they have, again, by subsequent inoculation reproduced the disease in monkeys in its clinical and pathological form.

Levaditi, Popper and Landsteiner have corroborated the findings of Flexner and Noguchi.

Pathological Investigations.

Ten *post mortem* examinations were made during the course of the epidemic. This represents a small percentage of the total deaths in Wellington during the period, but is partly accounted for by the fact that in the early stages of the epidemic no pathologist was available to carry out the examinations. Appended hereto are briefs of typical cases examined.

CASE I.—B.B., female, *ætatis* eleven years. Admitted to Wellington Hospital on December 28, 1924, with a history of sudden onset of backache, headache and inability to swallow, stiffness of neck and flexors of thighs. Patient was unable to speak properly and had a measles-like rash on the extensor surface of arms and legs. There appeared to be a paralysis of the soft palate. In the afternoon the child developed respiratory embarrassment of a jerky nature. Two days later the breathing was very shallow and purely diaphragmatic. An acute erythema of the nose appeared, almost in the nature of a cellulitis. On January 1, 1925, the child became cyanosed and drowsy, with weakness of the arms and died on January 3, 1925.

Post mortem examination done the next day showed intense congestion and œdema of the brain. On section the grey matter of the cord was picked out as a deep red area, more marked in the cervical enlargement. There were areas of congestion and hæmorrhage in the floor of the fourth ventricle. The substance of the cord was soft and pulpy and bulged through the cut membranes. There was little naked-eye change in the other organs beyond intense congestion of the lungs which showed at the left base an area of collapse. Microscopical sections were prepared from the tissues of the central nervous system. There was nothing to be seen in the cortex and cerebellum beyond œdema and congestion. In the medulla and both cervical and lumbar enlargements of the cord there were areas in which the congestion of the vessels was very marked. There were tiny thrombi in some of the vessels and one area of the cord showed marked unilateral hæmorrhagic softening with round-celled infiltration of the grey matter and absence of nerve cells. There were milder lesions on the opposite side with well marked perivascular collars of lymphocytes and plasma cells. In the floor of the fourth ventricle there was a unilateral hæmorrhagic area with round-celled infiltration.

CASE II.—W.W.C., male, *ætatis* two years. Admitted on January 4, 1925, with a history of four days' drowsiness, rash on body and loss of power in left arm and loss of superficial and deep reflexes. Spinal fluid was slightly under pressure, was faintly opalescent and contained twenty-five cells per cubic millimetre. On January 5, 1925, the child had convulsions, the respirations became sighing and irregular and nystagmus was now present. The child was listless and died on January 6, 1925.

Post mortem done the next day showed the usual deeply congested brain and cord with hæmorrhagic areas in the grey matter of the cord, most marked in the left arm region. Lungs were congested and œdematous.

Microscopically the cortex showed nothing of note. In the medulla there were numerous cellular lesions similar to those described above. In the cord there was marked round celled infiltration affecting the meninges with congestion of the meningeal vessels. Numerous perivascular collars in the grey and white matter of the cord.

CASE III.—B.J., female, *ætatis* eight years. Admitted on December 29, 1924, with a history of several days' malaise, slight cough and inability to stand. The child presented bilateral intercostal paralysis and both legs at this stage appeared to be spastic. There was difficulty in swallowing. Knee jerks and abdominal reflexes were absent and harsh rhonchi were heard over both lungs. On January 6, 1925, the child became comatose and developed a weak cough. Died next day.

Post mortem done the next day showed the same congestion and œdema of the brain and cord. There were naked-eye lesions in the cervical region on both sides and in the upper thoracic region. Both lungs were the seat of a septic broncho-pneumonia, most marked at the bases.

Microscopical findings were hæmorrhages, thrombi and softening with diffuse round celled infiltration. There is bilateral lysis of the anterior horn cells, the remainder are indistinct in outline as to cell and nucleus. There is congestion and œdema of the posterior columns.

CASE IV.—P. W., male, *ætatis* eleven years. Admitted on January 12, 1925, with a history of three days' feverish headache and inability to walk. He presented a complete intercostal paralysis and paresis of all limbs with difficulty in swallowing. Passive flexion of the neck was painful. Spinal fluid was opalescent and under slight pressure. Slight increase in globulin and six cells. Knee jerks were absent. A bulbar paralysis appeared and progressed rapidly, death occurring on January 13, 1925.

At *post mortem* we noted congestion of the brain which was very marked in the floor of the fourth ventricle in which situation there were also petechial hæmorrhages. In the lumbar region of the cord there was a nodular area of softening. There were hæmorrhages in the lumbar and cervical enlargements and less markedly in the dorsal region. The lungs were congested and showed numerous interstitial hæmorrhages.

The microscopical findings were in every way identical with those of the case described above.

CASE V.—D.R., male, *ætatis* eight years. Admitted on January 11, 1925, with a history of two days' frontal headache and pain in the back of the neck with vomiting and thickness of speech. There was difficulty in swallowing. The child presented a dusky flush and there was paresis of left face and palate. The speech was thick and nasal and the child could only swallow slowly. Spinal fluid was slightly opalescent with seven cells per cubic millimetre. The child's condition became gradually worse until the sixteenth, when it developed slow gasping respirations and weak pulse and died on January 18, 1925.

Post mortem done next day showed extreme congestion of the brain and cord with intense œdema. There were punctate hæmorrhages in the medulla one millimetre below the floor of the fourth ventricle. The cervical region of the cord is more deeply congested than the rest.

CASE VI.—L.R., a male, *ætatis* three years. Admitted on January 15, 1925, with a history of a two days' feverish head cold, followed by one day with pain in the back and weakness of the legs. The child was irritable, had a meningeal cry and a double cervical adenitis. Both legs were paralysed. There was marked cervical rigidity and the deep reflexes were absent. Spinal fluid was opalescent and under slight pressure. There was a definite increase in globulin and there were one hundred and twenty-six cells. On January 17, 1925, both arms became paralysed and the following day intercostal paralysis developed with rapidly ascending paralysis and the child died comatose on January 18, 1925.

At *post mortem* the next day there was general congestion and œdema with several extensive hæmorrhages into the floor of the fourth ventricle. The whole of the grey matter of the cord in all areas was disorganized with hæmorrhages and œdema. The cord was pulpy and soft and bulged through the membranes when cut across. The lesions were most marked in the lumbar region. The lungs showed hæmorrhagic spots.

Microscopical examination of the cord shows that the meninges are thickened and there is marked infiltration of the anterior horn with round cells. In the same site there are many hæmorrhages and some fairly large vessels show thromboses. There were poorly marked round cell perivascular collars. There is extreme dilatation of the capillaries of the grey matter. There is marked lysis of the cells of the anterior horn. There were marked perivascular lesions in the posterior columns. There was marked congestion of the liver with round celled infiltration in the portal tracts.

CASE VII.—C.B., male, *ætatis* eight years. Admitted on January 31, 1925, with a history of having been ill seven days previous followed by two days' remission and then four days ago an attack of headache, backache and vomiting, followed by another two days' remission. This morning he vomited and was feverish and sweated freely. He had a papular rash on the abdomen, cervical rigidity with tenderness and bilateral Kernig. Spinal fluid was cloudy, with much increase in globulin and six hundred and seven cells per cubic millimetre. On February 1, 1925, weakness of the arms appeared with pain in the left arm and respiratory distress and complete paralysis of the diaphragm. There was marked tachycardia. Child died on February 2, 1925.

Post mortem done the next day revealed marked congestion of the meningeal vessels. There was a cortical cyst, the size of a pigeon's egg, in the right parietal region. There was a small subependymal hæmorrhage in the floor of the fourth ventricle. The cord was tense with œdema and the cut curface bulged through the membranes. The grey matter of the anterior horn, especially in the lumbar and cervical enlargements, was irregularly darkened and stands out prominently.

Microscopically there is engorgement of the meningeal vessels with some degree of diffuse round celled infiltration and general congestion and œdema of the cord, with lysis of some of the anterior horn cells. There are engorged and thrombosed vessels with perivascular collars. There are similar lesions in the posterior horns. The medulla throughout all parts show perivascular round celled infiltration.

CASE VIII.—J.H., male, *ætatis* seven years. Admitted on December 8, 1924, with a history of seven days' illness with feverishness, pain in the abdomen and vomiting. On admission the child was restless, sweating freely and had difficulty in swallowing. There was paralysis of the left deltoid with loss of knee jerks and abdominal reflexes. There were harsh rhonchi in the chest. On December 9, 1924, child became suddenly cyanosed with weak pulse and died. Spinal puncture yielded no fluid.

After my arrival in Wellington I was handed portions of cortex, cerebellum and medulla, of which I made microscopical examination. In the medulla there were engorged thrombosed vessels showing a well marked perivascular infiltration with lymphocytes and a diffuse round celled infiltration of the tissues. The findings were typical of poliomyelitis.

From the examination of the above cases one can only corroborate the findings of those who have previously described the disease. The most striking feature was the intense œdema of the cord which was always present. On removing the laminæ one often noted an œdematous condition of the extradural fat. On palpation it was always noticed that the cord was tense and indeed in some cases the consistence was that of india rubber. On transverse section of the cord the white matter bulged through the cut surface. The grey matter was almost always swollen so as to occupy a relatively much greater proportion of the cord section. In keeping with the degree of œdema present there was a corresponding congestion of all parts of the cord. In the grey matter there were often in addition hæmorrhagic areas, with the result that the grey matter stood out as a reddish or reddish-brown area. These hæmorrhagic areas were seen in the medulla in the majority of cases examined. Microscopically such areas showed dilated vessels in some cases thrombosed and some with diffuse areas of hæmorrhage in the vicinity. The cellular areas were of great interest. In the perivascular lymphatics in the engorged areas were noted collar like collections of round cells with deeply staining nuclei. In none of the sections examined did we notice polymorphonuclear cells in the inflammatory cells in any situation. In addition to these lesions were noted commonly diffuse round celled infiltrations of the grey matter without any particular relation to the blood vessels. In the more severe cases these cells were aggregated into clumps and in some few cases were disposed round the central canal of the cord. Only in a few cases did we note an inflammatory infiltration of the meninges and when this did occur, it had no special distribution with regard to the vessels. In none of the cases did we note any infiltration of the meninges of the brain. In cord and medulla we encountered areas in which softening had occurred and in many cases this involved the white matter and microscopical examination revealed numerous vacuolated spaces. In a small proportion of the cases examined the naked eye appearances were quite similar to the typical cases described above, but on microscopical examination no cellular lesions were found. There was, however, extreme congestion of the capillaries of the grey matter and tiny multiple extravasations of blood round these engorged vessels. These cases were noted clinically as definite cases of the disease, but following an extremely rapid course. The interpretation placed on these findings was that the overwhelming virulence of the infection left no opportunity for tissue response.

Bacteriological Investigations.

Cultures and subcultures were made of material from the central nervous system of four definite clinical cases of poliomyelitis which terminated fatally, and cultures were also made in a similar manner from two definite clinical cases of experimental poliomyelitis in monkeys (*Macacus rhesus*).

The medium employed in almost all cases was that suggested by Noguchi, that is, fresh sterile ascitic fluid in which was placed a fragment of kidney from a rabbit or a guinea pig. The kidneys were removed aseptically from freshly killed animals. The medium was incubated overnight and tested next day for sterility; any contaminated tubes were rejected. The temperature of incubation throughout was 37° C. and the conditions were anaerobic. Anaerobiasis was maintained by placing a layer of sterile paraffin on the surface of the medium. Some were in addition placed in airtight jars from which the oxygen was removed by the combined action of exhaustion and pyrogallol. The filters were Chamberlain F. and a very fine type of Muencke porcelain candle. Using these filters for a great variety of bacterial growth, it was evident that no ordinary bacteria were able to pass. Throughout the experiment every step was checked by means of controls.

The first lot of material to be cultured was from Case V. A portion of the cord, taken from a segment in which the lesions were well marked, was removed at *post mortem* and planted in a tube of the medium. After five days' incubation very small bluish bodies were seen in smears stained with Giemsa. These were, however, present in the control tube, though the arrangement was not so regular in the latter. The culture was then filtered through a Chamberlain F filter and subcultured on similar medium. After five days' incubation of the primary culture an opalescence of the medium gradually occurred. This was also present in the control and was probably due to autolysis of the kidney and cord tissue.

The next cultures made were from Case VII. The cord and brain were flamed and four primary cultures were made. After five days there was no result, except that some purple staining bodies were seen with Giemsa both in cultures and controls. A 5% emulsion of brain in sterile water was also made and put through a filter. Four cultures were made from this, but in five days there was no visible growth.

The next case examined was that of a child at Wanganui who had died during a typical attack of the disease. At the request of the Department I went to Wanganui and made a *post mortem* examination in this case. The cord was removed and portions kept in 50% glycerine, the

remainder was packed in ice in a "Thermos" flask. (Used next day for animal inoculation, *quod vide*.). The glycerinized cord was kept for five days, after which three pieces were planted in medium. The medium in this case consisted of equal parts of ascitic, pleural and hydrocele fluids in which was placed one gramme of sterile rabbit kidney. All cultures showed after five days a streptococcus and a Gram-negative bacillus, also minute organisms (? cocci) in clumps and chains, very much smaller than the streptococcus with which they were growing. It was noted that the streptococcus was markedly Gram-positive, whereas the smaller bodies did not retain the stain by Gram's method. These cultures were then passed through a filter and subcultured without anything definite being found.

The next material for culture was from a monkey, B in our series, which was killed with chloroform during the acute stage of the disease. The cord was carefully removed and planted fresh in the medium. After five days a smear was made and stained with Gram. In it were noted Gram-negative bacilli, Gram-positive bacilli and tiny "coccoid bodies" in clumps, short chains and pairs. Some of these clumps were surrounded by clear zones. The small bodies observed appeared to be about 0·1 μ in diameter. These cultures were cultured and subcultured. After five days smears showed similar bodies in pairs only which stained well with Giemsa and did not resemble the granules in the control tube which did not stain so definitely. A subculture was also made without filtration and this showed a profuse growth of these small bodies, much heavier than in the primary culture. In these subcultures contaminating organisms also flourished, but there were no contaminating organisms in the cultures from the filtrate. These small bodies we regarded as the "globoid bodies" described by Flexner and Noguchi. Direct smears made from the brain of the same case when stained with Giemsa showed small bodies with indefinite outline, but a control from a normal guinea pig brain showed similar bodies which were thus regarded as being non-organismal. Three lots of streptococci growing in primary culture from this monkey's brain were cultured under anaerobic conditions for five days. In two tubes the cocci remained the same size without marked pleomorphism. A smear from the third tube revealed many small forms all of which were, however, much larger than the bodies above mentioned and all of which were markedly Gram-positive.

Cultures were also made from the medulla of monkey B on solid media. This medium was prepared in plates as described below; the medium was inoculated and incubated anaerobically in a Buchner jar. With a two-third inch lens many dew-like colonies were seen after five days' incubation, lying in close proximity to the colonies of contaminating organisms. Some appeared to be growing on top of the larger colonies. Attempts were made to inoculate Noguchi medium with these with only partial success, as none of the subcultures were pure. Possibly, we thought, the globoid bodies were growing in symbiosis with the other organisms.

Portion of the cerebrum of Monkey E (experimental poliomyelitis) was removed with all possible precautions and planted in Noguchi 3F medium. In ten days a portion of brain was removed and crushed out; this showed with Giemsa contaminating organisms and numerous globoid bodies. Portion of cerebrum, lumbar cord and medulla treated fifteen minutes with 2% phenol, washed in saline, were planted in medium 3F. The results were in every case similar to the above.

Media.—Noguchi A medium was made and used as follows: Untreated ascitic fluid was filtered through a Chamberlain F filter and tubed in ten cubic centimetre quantities to which was added about one gramme fragments of sterile guinea pig kidney; these were tested for sterility and covered with one and a half inches of sterile paraffin. Noguchi 3F medium was made from a mixture of equal parts of hydrocele, ascitic and pleural fluids, filtered, tubed in ten cubic centimetre lots and to this was added sterile rabbit kidney in one gramme pieces overlain with one and a half inches of paraffin. Solid medium 3FA: One rabbit's kidney was ground up with sand and ten cubic centimetres of distilled water and triturated for about twenty minutes. This was filtered through filter paper and then through a candle. The filtrate was now added to thirty cubic centimetres of 3F medium above and the whole filtered through a sterile Muencke filter. To each volume of this filtrate was added one volume of agar of reaction + 0·6 (Eyre) at a temperature of 40° C. and poured in plates.

From the above observations it will be seen that the globoid bodies described by Flexner and Noguchi were observed in the cultures from the cases in both human and experimental poliomyelitis. We were not able to overcome the difficulties of growing the organism in pure culture. The frequent gross contamination with other organisms over the long periods required for incubation prevented us from carrying the cultures through many generations of subcultures.

Experimental Investigations.

In commencing this work we realized that little could be done until we had produced experimental poliomyelitis. Consequently we obtained a small supply of monkeys from the municipal authorities at Wellington and Auckland. As previous experience seemed to show that the intracerebral method of inoculation gave the best results, we decided to use this method exclusively. Through the kindness of Dr. Wilson who gave us every possible assistance, suitable accommodation was got for the monkeys at the Hospital. We were also given part of the Out-patient Department and such theatre equipment, gowns *et cetera* as were necessary to carry out the operative work under aseptic conditions.

The first case used for experimental purposes was Case VI. in the series above. On January 22 a 20% emulsion was made from glycerinized cord. Dr. Wilson anæsthetized monkey A with ether and after shaving and sterilizing the scalp, dressing sheets were put in position and a skin and muscle flap was turned down in the parietal region. A half inch trephine opening was made, leaving the dura uninjured. With a 24-gauge needle 0·2 cubic centimetre of emulsion was injected deeply into the brain substance. The skin and muscle flap was sutured back in position. After placing a collodion dressing over the wound the monkey was returned to its cage. It rapidly recovered from the operation and seventy days after showed no signs of paralysis.

Monkey B was inoculated in exactly the same way on January 31, 1925, except that a quarter inch trephine opening was used; 0·4 cubic centimetre of a fresh emulsion (20%) of cord (Wanganui case) which had been kept on ice for twenty-four hours, was injected through a trephine opening in the parietal region. On February 11, 1925, it was noticed that the animal was shivering. On the morning of the next day the animal was obviously ill and feverish, eyes were running, refusing food, unsteady gait and loss of interest in surroundings. In the afternoon the condition was worse, the lower jaw was inclined to the right and there was some weakness in the hind legs. On February 13, 1925, the animal lay on its back unable to move and its respirations were slow. Temperature normal. Examination showed pupils equal and reacting to light. No strabismus. Corneæ slightly glazed. No facial paralysis. There was evidently difficulty in swallowing. Forcible flexion of the head gives the animal pain. Both arms are paralysed, the right was flaccid and the left slightly spastic. Deep reflexes lost. There was paralysis of the intercostals. Abdominal reflexes present. There was now some paresis of the legs, but knee jerks were brisk. February 14, 1925: Respiration twenty-nine. Right leg was flaccid with absent reflexes. Paralysis of arms is now of flaccid type on both sides. Abdominal reflexes were now absent. February 16, 1925: Spinal puncture fluid not under pressure. Five cubic centimetres withdrawn. Globulin greatly increased, thirteen cells per cubic millimetre. Animal was now *in extremis* and was killed with chloroform.

Post mortem: The brain showed œdema and marked congestion, the vessels in the floor of the fourth ventricle were congested, but there were no hæmorrhages. The cord was tense with œdema and the meningeal vessels were

injected. On section of the cord there were marked lesions in the grey matter of the cervical region, less in the dorsal and indefinite in the lumbar region. The operation wound was soundly healed and there was no evidence of hæmorrhage or infection at the site of inoculation. The mesenteric glands were not palpable.

Microscopical examination of the cord showed precisely the same lesions as were noted in the human cases. The perivascular collections of small round cells were to be seen most marked in the cervical region. There were no lesions in the meninges of the brain at the site of inoculation or at any other part.

Monkey C was inoculated intracerebrally on February 2, 1925, with 0·2 cubic centimetre of a 20% emulsion of the cord of Case VII. At the termination of the operation the animal collapsed and could not be brought round.

Monkey D was inoculated in the same way and with the same quantity of virus from Case VI. Six days later the animal appeared to be seedy with a tendency to drag the left leg, but this passed off and monkey recovered completely.

Monkey E was inoculated with emulsion of cord from a case which was at the time believed to be poliomyelitis, but which on subsequent examination proved to be a case of liver necrosis. On February 16, 1925, one week after the above inoculation, the animal was reinoculated through the same trephine hole with an emulsion of cord from monkey B which had had the disease. February 24, 1925: The animal was going off its food and two days later it was seen crouching in the corner of its cage, shivering, with its head pressed against the wall. There now appeared some difficulty in mastication and swallowing. February 27, 1925: Feverishness had now gone, but the animal was much worse. Mastication was impossible and animal had to be fed with a pipette. The back legs seem very weak. February 28, 1925: There was now in addition definite stiffness of the neck, paralysis of the left deltoid, paresis of both arms, intercostals working feebly, both legs flaccid paralysis and deep reflexes lost. March 2, 1925: There was some slight recovery in the left foot. March 3, 1925: Respirations were much more difficult, the intercostals have only slight movement, right arm alone unaffected. March 6, 1925: Paralyses remained unchanged. Spinal puncture showed fluid not under pressure, with a slight increase in globulin and nine hundred cells per cubic millimetre. Animal killed with chloroform.

Post mortem showed that the site of inoculation was soundly healed. There was marked congestion of the cortex. The meninges of the cord were congested and the cord itself was tense with œdema. There were no macroscopic lesions in the medulla, cervical or dorsal regions. Lumbar enlargement showed swelling and darkening of the anterior cornu. The mesenteric glands were enlarged and soft. There were some recent adhesions in the left pleura. There were no naked-eye changes in any other organs.

Microscopical examination showed the same appearances as seen in other cases except that there appeared to be some organization and the formation of young fibrous tissue in the anterior horn. No further inoculations were made with this material. It will thus be seen that of five monkeys inoculated there were two definite cases of the disease, proved clinically and pathologically. Further that although from these cases we had a source of fresh virus which could immediately after death be placed in culture media, we had the very greatest difficulty in cultivating the virus and failed completely to grow it in pure culture. Twice at the request of the Department I visited Auckland and worked on the same lines in conjunction with Dr. Gilmour. Ample material was available for culture purposes and well over a hundred cultures were prepared very carefully, without producing a growth of anything resembling globoid bodies. This, of course, made the preparation of a vaccine or an immune serum a technical impossibility.

With a view to repeating the findings of Rosenow we inoculated intracerebrally through trephine openings thirteen rabbits. We used 20% emulsions of infected cords, injecting 0·2 cubic centimetre in each case. Five rabbits died from septic meningitis or some other cause related to the operation itself. None of these showed any lesions suggestive of poliomyelitis, although some developed paralysis of the hind legs before death occurred. This was, however, found later to be the case with rabbits which had been inoculated with streptococci of low virulence from various sources and has no relationship anatomically or bacteriologically to that occurring during an attack of poliomyelitis in a man or in a monkey.

Of the rabbits that recovered from the operation, none developed anything suggestive of poliomyelitis. We came to this conclusion after examining microscopically the cords of these animals after death.

We regret that out of the volume of work done in connexion with the epidemic, nothing has arisen which is at all likely to be of assistance from a therapeutic standpoint. Attempts were made to estimate susceptibility by skin tests. Dr. Frengley suggested the use of an extract of proteins from the spinal fluid of infected cases. This was found to be not readily carried out, but, acting along the lines of his suggestion, the following method was used: Cord from a case recently dead was made up to a 20% emulsion with saline by grinding in a mortar. This was then filtered through a porcelain filter; the filtrate was heated to 56° C. for forty-five minutes. About one hundred children in the ward of the Auckland Hospital were tested by intradermal injection of 0·2 cubic centimetre of this filtrate. Half of this number were children who were ill with or convalescent from poliomyelitis. In no case was there the slightest reaction which could not be attributed to the mechanical injury to the skin. This destroyed any hopes we had of being able to devise some test on the same principle as the Schick test. I have now made arrangements to hand what cultures and glycerinized cords we have to Dr. Hercus who is making arrangements to carry on the work. It is my intention to visit Dunedin during the next week and explain to him the details of the work, as this has for the most part been described in outline only in the above report.

I am very grateful for the assistance afforded me by the Department. Everything possible was done to facilitate the work, both in Wellington and other places that I visited for the purpose of collecting material.

If there are any points which you may think are not covered by this report, I should be very pleased to supply them from such notes of the work as I have kept.

Yours respectfully,

April 4, 1925. P. P. LYNCH, M.D.

Means of Spread.

Nothing new was discovered. In the Wellington Health District, however, the evidence of spread by personal contact is exceptionally strong. I append extracts from a report by Dr. R. A. Shore, Medical Officer of Health, which demonstrate this.

The present epidemic may be said to have put in an appearance during the last week of November, 1924, the first case being notified on November 25, 1924, from Wellington City. Two more cases were notified before the end of the month, one from Wellington City and one from Lower Hutt. During December the disease waxed apace and except for one instance all the cases were notified from Wellington City and its immediate surroundings.

Of the total of fifty-six cases notified in the Wellington Health District up to the end of December, 1924, fifty-five of them occurred in Wellington City and surrounding area. One case was notified from Napier. Inquiry, however, showed that this child had only two or three days previously gone to Napier from Wellington, so that during the last week of November and all December the epidemic confined its attention to the Wellington City and surrounding districts.

One fact impressed one while making inquiries as to the possible source of the epidemic and that was that during November there was amongst the children a considerable amount of mild indefinite illness, variously diag-

nosed as influenza or summer sickness and characterized by fever, malaise and drowsiness.

Epidemic in January, 1925.

With the advent of holiday traffic and consequent greater number of people moving from the city to the country and much greater personal contact occurring between people of different districts, the month of January soon showed that the epidemic was going to cover a much greater tract of country, so that in considering the course of the epidemic in January a much wider area comes under review.

As in December, a large number of cases still occurred in the old area. More suburbs were invaded, as for instance, Kelburn, Khandallah, Johnsonville, Roseneath, Wadestown, Thorndon, Hataitai, Ngaio, so that by the end of January, 1925, it may fairly be said the whole Greater Wellington area had become infected. The incidence of cases in the suburbs first infected, namely Lower Hutt and Petone, showed a considerable falling off.

Outside of the Central Wellington area the first case was notified from Gonville on January 3, 1925. This patient (Case 6/25) left her home in Brooklyn, Wellington, on December 22, 1924, and stayed in Wanganui until December 27, spending the whole day on December 25, 1924 (a public holiday), on the beach at Castlecliff. On December 27 she first complained of pain in back of neck. The illness was rapidly progressive and she died on January 3, 1925. In connexion with this case it is worthy of note that the householder is manager of a picture theatre, another inmate is ticket seller at the same place and that child during incubation period spent a day on the beach at a time when the beach was crowded with other children and also that the day on which she first complained was spent at Stratford. So that even with the first case in Wanganui District there were undoubtedly numerous contacts.

The two following patients from Wanganui had also spent considerable time at Castlecliff beach during the Christmas and New Year holidays. The fourth patient was a direct contact with the second patient and is rather interesting. This patient (31/25) had been in hospital under treatment for a broken arm and discharged on December 20, 1924, and went to reside at Castlecliff and spent some hours on the twenty-sixth playing with patient 11/25 who was his cousin and first complained on January 6, 1925. Recovered apparently and again became ill on January 11, 1925. Was removed to hospital and now has paralysis of right arm.

The next patient in Wanganui was probably also the innocent means of further spreading the infection. His source of infection was probably a public picnic that he attended on New Year's day. First symptoms developed on the tenth, but that same evening in spite of the fact that he was not feeling well he attended a junior meeting of a religious character. Next day was ill and was under treatment for "sunstroke" until the thirteenth, when some paralysis appeared and he was put in isolation at hospital.

So with these first four cases occurring in Wanganui and coming into direct contact with so many other children, it could only be expected that Wanganui and district should be rather extensively affected. The subsequent course of the epidemic in that district quite proved that surmise. Quite early in the epidemic in this district there were also other lines of contact from Wellington.

Masterton and District.—The first notification from Masterton and district was received on January 9, 1925, an adult woman, age twenty-eight years (Case 18/25). At first efforts made to trace the source of infection in this case met with no success. The case occurred at an isolated country centre a considerable number of miles from Masterton and no contact with any outside source could be established. However, more minute inquiries conducted by two special officers of the Department cleared up the mystery and a direct source of infection from Wellington could be traced and in addition the fact that there had been a fair amount of so-called "summer sickness" amongst the children in this district was elicited. This summer sickness could be traced directly back to Masterton, between which town and Wellington there is extensive communication.

However, having once reared up its head the epidemic soon grew and spread throughout the whole district of Wairarapa.

Palmerston and District.—The first notification from Palmerston and district was on January 10, 1925, an adult man, age fifty years, who was ill for one week before advent of paralysis which ultimately affected both legs. This district showed evidence of two distinct avenues of infection, one from Wanganui and one from Wellington. The second patient notified, who was really the first case in the district, had visitors from Wellington and Wanganui. The third patient also gave history of having had visitors from Wellington at New Year time.

A special word is perhaps required with regard to the Levin area of this district. Early in the epidemic here (January 3) a birthday party was given. At the birthday party there were children present from Auckland, Wellington and Masterton, besides local children. About ten days later all the children of this family, eight in number, suffered from an illness called "summer sickness" and one of them developed infantile paralysis and died on January 18, 1925. This child while in the incubation period, in fact she had her first symptoms in the train, travelled from Levin to Wanganui and before arrival at Wanganui was showing signs of muscular weakness, "as she walked she dragged her legs." So here was a patient with infantile paralysis travelling in the train during the most infective period of the disease and coming freely in contact with other people and at one station visited the refreshment rooms.

So taking into consideration these facts it is not surprising that Levin district showed a large number of notifications. A point to be noted in a great many of these cases is that the various beaches were evidently a starting point for spread of infection as in numerous cases we get history of the patients having visited the beaches and this at a time of the year when these seaside resorts might reasonably be presumed to be more than normally densely populated.

New Plymouth and District.—The first case notified was from Waitara on January 12, 1925. This patient (29/25) travelled from Inglewood to Waitara on January 8 and then moved on to Motonui and was notified on the twelfth after four days' illness. The report submitted on this case states that a visitor from Wellington arrived on January 3 and that this visitor gave a history of having had recent gastro-intestinal symptoms with headache and lassitude. Waitara also had a case of infantile paralysis in September, 1924. This case had a fatal termination and it is also of interest to note that most of the cases in the New Plymouth district came from Waitara.

The total number of cases notified during January was 143.

February, 1925.

February, 1925, showed a total notification of 163 cases, showing a wider distribution, practically general over the whole Wellington Health District, except that the district north of Woodville and the Hawke's Bay district still showed a considerable immunity, only four cases being reported through this whole district in the month of February.

Wellington City area showed a decrease, only thirteen cases being notified during this month.

Wanganui, Palmerston and Masterton districts still showed evidence of the active presence of the disease, the epidemic now having radiated from these centres into the smaller centres and surrounding country districts.

The following case histories will serve to show the mode of spread of infection:

Case 174/25: The patient was a female, aged nine months. Illness commenced on January 26, 1925, but was not finally diagnosed until February 3, 1925, when some paralysis of the muscles of the trunk was observed. During this period when the child was under observation her

cousin (Case 214/25) age two years, was in close contact, sometimes leaning over the child's go-cart to play with her. This second child was notified as suffering from poliomyelitis on February 8, 1925, and on following day (February 9, 1925) her father's condition was also notified as anterior poliomyelitis.

Case 87/25: The patient was a female, aged two years. Her condition was notified as a case of poliomyelitis on January 19, 1925, and subsequently she developed paralysis of both legs. Patient 116/25 was in close contact with this girl up to January 18, 1925, and was notified as a definite case on January 25, 1925.

Case 51/24: The patient was a female, aged eleven years. Initial symptoms appeared on December 24, 1924, and were quickly followed by a rash and general weakness, inability to swallow or to speak distinctly. The rash was general and of a "measly" character. The child died on January 5, 1925, from bulbar paralysis. On December 28, 1924, four days after the onset of disease in the girl, a brother, aged fifteen months, developed initial symptoms of infantile paralysis and was subsequently diagnosed as a definite case and removed to hospital and ultimately recovered without paralysis. Another child, female, aged ten years (Case 48/24), who had been in close contact with both of the above, was notified as a definite case on December 29, 1924, and subsequently had developed bulbar paralysis and died on January 3, 1925. Another child who had been staying as a visitor at the home of 51/24 and returned home, was under treatment for an illness with symptoms pointing to a mild attack of infantile paralysis.

On December 26, 1924, a girl, aged one year and five months (Case 53/24), developed an illness with following symptoms, high temperature and severe pain in back and neck. On December 27 and December 28 the condition improved, but on December 29 was much more seriously ill and removed to hospital, where death occurred on January 5, 1925, as result of bulbar paralysis and bronchopneumonia. On January 4, 1925, a boy, four and a half years (Case 8/25), developed an illness with following symptoms, pain in the back of the neck, drowsiness, irritability and vomiting. He was removed to hospital and subsequently had some paralysis of both legs. The families of these two cases are relatives of each other and were in close and daily contact from commencement of the illness of 53/24 until her removal to hospital.

Case 126/25: A boy, aged eight years, became ill on January 21 and finally diagnosed as anterior poliomyelitis on January 26, 1925. The history showed that this boy had been in close contact with a case removed from same neighbourhood and that they had been constantly playing together.

Case 170/25: A boy, aged thirteen years, was a close contact of patients 116/25, 135/25 and 136/25, the whole three cases occurring in one family.

Case 275/25: A girl, aged two years and seven months, notified on February 14, 1925; was direct contact with 160/25 which was notified twelve days previously. Patient 275/25 developed first symptoms on February 9, 1925, so that period between exposure to infection and commencement of illness was seven days.

Case 83/25: The patient was a female, aged two years. Her condition was notified as anterior poliomyelitis on January 20, 1925. This family was visited on January 19, 1925, by a lady who then proceeded direct to her sister's home to assist in attending to the baby. This baby (Case 130/25) was notified as a case of poliomyelitis on January 27, 1925. All the children of the family of patient 130/25 were prior to onset of the illness habitual playmates with another family X. When the household of which patient 130/25 was a member, was put into isolation, the family of X then played with another family of children and on February 5 Patient 200/25, a member of the last family, became ill and was notified on February 9, 1925. Case 82/25 was a severe case and the patient had paralysis of both legs. Case 130/25 was a severe one and the patient died on January 29, 1925. Case 200/25 was a severe case and the patient had intercostal paralysis.

Case 37/25: A boy, aged three years, became ill on January 10, 1925, and his condition was definitely diagnosed as anterior poliomyelitis on January 25 and he died on January 21. On January 12 a cousin of above (Case 57/25) became ill and was removed to hospital on the sixteenth as a case of anterior poliomyelitis. The family of the latter was visited by a lady a week previous to January 15 and she then returned to her home and on January 12 her daughter (Case 56/25), aged ten years, developed an illness which was diagnosed on the fifteenth as infantile paralysis, and developed right facial paralysis and paresis of the right arm and leg.

Case 11/25: A male, aged eight years, was spending holidays at Castlecliff (where first Wanganui case originated, having come from Wellington) and his condition was diagnosed as anterior poliomyelitis on January 5, 1925, with paralysis of intercostal and *rectus abdominis*. On December 20, 1925, patient 31/25 arrived at Castlecliff and visited family of 11/25 at intervals from December 26, 1924. Patient 31/25 was first "seedy" on January 6, 1925, and after a temporary recovery showed further symptoms on January 11, 1925, was removed to hospital and developed paralysis of left arm.

Further cases could also be quoted, but it would seem that the above present sufficient evidence to show that not only are cases developed from contact with actual patients both before and during the onset of illness, but also that infection can possibly be carried by an apparently healthy person who has come into contact with an actual case.

Interesting Points in the Symptomatology.

In going through case reports there are one or two rather interesting features that might well be mentioned, although it is not intended to go extensively into the symptomatology of the disease, still the following points may be of interest.

Occurrence of Convulsions.—Occurrence of convulsions was rare and in the 365 cases investigated up to the end of February, 1925, there were only seven patients that gave a history of either one or more convulsions as an initial symptom or only 1·91% of cases.

Occurrence of a Rash.—There were eight patients, or nearly 2·2% that gave a history of having a "rash." There was nothing typical or uniform about either the appearance or distribution of the rash.

Patient 51/24 had a general rash of a "measly" character.

Patient 5/25 had a general "spotty" rash.

Patient 124/25 had a rash (not described) limited to the region of the wrists and knees and perioral area.

Patient 130/25 had rash on head.

Patient 142/25 had rash on the neck.

Patient 180/25 had general scarlatiniform rash.

Patient 218/25 rash on the face.

Patient 327/25 had a finely mottled rash on the chest and arms.

The occurrence of a rash would seem to be of serious prognostic significance. Of the eight patients six or 75% died and the other two developed serious paralysis.

Occurrence of a Period of Apparent Recovery or Remission of Symptoms.—The so-called hump-backed or dromedary type of case is fairly common. As far as could be judged from the reports of the cases it occurred in about 10% of the cases, but is probably more common as, unless the inspectors were specifically instructed to inquire about it, the occurrence of a remission of symptoms would be apt to be overlooked by the parents.

The length of the remission of symptoms varies from one day to six days, the most usual time being two to three days.

One patient showed a double remission of one day's duration in each instance.

Correspondence.

THE INTERMEDIATE HOST OF FASCIOLA HEPATICA.

SIR: I regret that my "Note on the Intermediate Host or Hosts of *Fasciola Hepatica* in New South Wales" in THE MEDICAL JOURNAL OF AUSTRALIA of March 13, 1926, should

have called forth so intemperate a letter from Dr. Bradley as that published in your issue of April 3. I am at loss to understand Dr. Bradley's extreme sensitiveness to any criticism of his article in regard to the water snails of Monaro and New England particularly since there was no "implied suggestion" as he apparently seems to imagine.

Dr. Bradley takes exception to the fact that in a note of this nature more details were not given in regard to the nature of the experimental animals used. I must admit that I left much to his imagination by my inadvertently referring to experimental animals instead of specifying laboratory bred guinea pigs. As to the description of the cercariæ and cysts, since such description and measurements are in themselves of little diagnostic value, there appeared no necessity to state them in a preliminary note.

It was to be expected that if Dr. Bradley was really actuated by a desire for knowledge and merely by the desire to give free vent to his personal feelings that he would at least have withheld his criticism until the detailed account of my observations was published, as was promised in the article he criticizes. As there were published in the *Journal of the Australian Veterinary Association* (March, 1926), just a few weeks after my contentious note, I trust he will have already found the evidence he so ardently desires.

Just what reason there is for reference to an interview between Dr. Bradley and myself, of which no record was kept, in what purports to be scientific comment, is hard to determine, especially since his memory of my remarks do not coincide with my own. How unlikely it is that I would have made such a remark as that attributed to me at the interview referred to, may be seen from reference to the distribution of *Limnæa brazieri* as recorded by me in the *Journal of the Australian Veterinary Association* (Volume II., No. 1). It is also difficult to perceive what grounds Dr. Bradley has for assuming that I have "profoundly altered" my views seeing that at the time of the above note there had been no previous publication of my opinion in regard to this matter.

Finally may I be permitted to hope that my alleged "ignorance" of Koch's "famous postulates" is no greater than that of Dr. Bradley's conception of the distinction between legitimate scientific criticism and mere personal abuse.

Yours, etc.,

A. C. McKAY.

The Veterinary School,
University of Sydney.
April 16, 1926.

Medical Appointments.

Dr. Clive Mansley Greer (B.M.A.) has been appointed Public Vaccinator at Warracknabeael, Victoria.

* * * *

Dr. Guy Austin (B.M.A.) has been apointed Acting Honorary Phyisician at the Adelaide Hospital, South Australia.

* * * *

Dr. Emanuel Sydney Morris (B.M.A.) has been appointed Director of Maternal and Baby Welfare, Office of the Director-General of Public Health, Sydney.

Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx.

QUEEN VICTORIA HOSPITAL FOR WOMEN AND CHILDREN, MELBOURNE: Medical Superintendent, Senior Resident Medical Officer.

THE PUBLIC SERVICE BOARD, NEW SOUTH WALES: Assistant Medical Officer of Health.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association.
QUEENSLAND: Honorary Secretary B.M.A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIAN: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Ceduna, Wudinna (Central Eyre's Peninsula), Murat Bay and other West Coast of South Australia Districts.
WESTERN AUSTRALIAN: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

MAY 4.—Tasmanian Branch, B.M.A.: Council.
MAY 5.—Victorian Branch, B.M.A.: Branch.
MAY 5.—Western Australian Branch, B.M.A.: Council.
MAY 6.—South Australian Branch, B.M.A.: Council.
MAY 7.—Queensland Branch, B.M.A.: Branch.
MAY 8.—New South Wales Branch, B.M.A.: Ethics Committee.
MAY 11.—Tasmanian Branch, B.M.A.: Branch.
MAY 13.—Victorian Branch, B.M.A.: Council.
MAY 13.—New South Wales Branch, B.M.A.: Clinical Meeting.
MAY 14.—Queensland Branch, B.M.A.: Council.
MAY 17.—New South Wales Branch, B.M.A.: Organization and Science Committee.
MAY 18.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
MAY 18.—Tasmanian Branch, B.M.A.: Council.
MAY 19.—Western Australian Branch, B.M.A.: Branch.
MAY 25.—New South Wales Branch, B.M.A.: Medical Politics Committee.
MAY 25.—Illawarra Medical Association, New South Wales.
MAY 26.—Victorian Branch, B.M.A.: Council.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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